Mr. Geoff J. Henley
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2520 Fairmount Street, Suite 200
Dallas, Texas 75201

January 10, 2023

Report of Martin J Tobin MD, Professor of Medicine,
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Dear Mr. Henley,

This report contains my opinions on the case of Vicki Timpa et al. v. Dustin Dillard et al; No. 3:16 -CV-03089-N. I have been retained by Henley & Henley, P.C., Dallas, and charges for my time are being donated to *Lamp for Haiti* charity. In reaching my opinions, I read the documents and watched the videos listed in Appendix #1. Appendix #2 contains a listing of timed events based on my viewing of the videos.

On Wednesday, August 10, 2016, Sergeant Kevin Mansell was assigned the case of Mr. Tony Timpa at 22:30:34 hours (Sergeant Mansell deposition). Sergeant Mansell arrived at the scene at 22:37, appended the call at 22:38:51, and team 533 arrived at 22:40. Five officers of the Dallas Police Department participated in the restraint of Mr. Timpa: Officer Dustin Dillard, Officer Danny Vasquez, Officer Domingo Rivera, Corporal Raymond Dominguez, and Sergeant Mansell. The timing of events listed in my report are not in actual real time but are instead based on the times of the Axon body camera of Officer Vazquez, which commenced recording approximately 38 seconds earlier than the body camera of Officer Dillard and 2 minutes 24 seconds before the body camera of Officer Rivera.

Pivotal elements and utterances in the timeline

00:51 Mr. Tony Timpa: “You’re gonna kill me. You’re gonna kill me. You’re gonna kill me”
01:27 Officer Dillard and Officer Vazquez turning Mr. Tony Timpa prone
01:28 Officer Dillard and Officer Vazquez have turned Mr. Timpa prone
Off. Dillard’s two hands and Off. Vazquez’s left-hand press on Mr. Timpa’s torso
01:29 Off. Dillard places his left knee on Mr. Timpa’s torso, and it remains on Mr. Timpa’s torso until 15:36 (with exception of 3-sec interval between 14:29 and 14:32)
Off. Vazquez places his left knee on Mr. Timpa’s torso, and it remains on Mr. Timpa’s torso until 4:06
02:14 Off. Dillard places his right knee on Mr. Timpa’s torso, and it remains on Mr. Timpa’s torso until 03:27
02:28 Paramedic visible in the frame (until 02:52)
03:02 Officer: “Don’t need to be squirming man”
03:10 Mr. Timpa: “Please leave my feet alone”
04:28 Off. Vazquez working on handcuffs
05:43 Cpl. Dominguez and Off. Rivera restraining Mr. Timpa’s legs
06:02 Officer: “He’s squirming way too much”
06:09 Officer: “Stop, Tony, stop. You’re going to have to stop squirming”
07:18 Mr. Timpa: “It hurts. Please take it off”

07:19 Handcuffs removed
07:26 Mr. Timpa: "My chest hurts"
08:38 Paramedic taking blood pressure [BP 150/90, P 100, R 20]
09:28 Off. Vazquez's left hand is on Mr. Timpa
09:36 Mr. Timpa: "Kill me my friend. Kill me. Kill me. I need to die"
10:25 Mr. Timpa: "I'm gonna die. Help me. Help"
10:39 Mr. Timpa: "You're gonna kill me. Oh God. Oh God"
10:45 Mr. Timpa: "I feel terrible. Help me. Help me."
10:50 Mr. Timpa: "Help me. Help me. Help me. Help me. Help me."
11:23 Mr. Timpa: "Help me. Help me. I don't want. Help me. Help me. I don't want"
11:36 Mr. Timpa: "Help me"
11:40 Mr. Timpa: "Help me"
11:55 Mr. Timpa stops lifting his head and extending his neck
12:01 Rhythmical motion of Mr. Timpa's head occurs repeatedly up until 13:33
13:00 Officer: "He was squirming a little too much"
13:23 Mr. Timpa's left arm moves
Officer: "Yeah, he's still breathing; he snorted"
13:29 Off. Vazquez's right hand is on Mr. Timpa
13:33 Rhythmical motion of Mr. Timpa's head stops
13:44 Officer: "Shit, if I was squirming that much I'd be sleeping too"
14:03 Off. Dillard shakes Mr. Timpa's left shoulder, preceded by vocalization of "Tony"
14:18 Off. Dillard vigorously shakes Mr. Timpa's left shoulder, preceded by "Tony"
14:40 Paramedic injecting Versed
15:36 Off. Dillard removes left knee from Mr. Timpa's torso
16:00 Mr. Timpa being lifted on to gurney

Vasquez Body Camera 01:39

Screenshot from Officer Vazquez's body camera at 01:39 (~10 sec after compression of Mr. Timpa's torso began). Officer Dillard's left knee is pressing down on Mr. Timpa's prone torso, with toe of left boot raised and can be seen to swing freely on video. Officer Dillard's

left upper extremity, with elbow in extension, is compressing Mr. Timpa's torso. Officer Dillard's left hand is on torso, although not fully visible in this frame (visible on closely related frames). On right of screenshot, Mr. Timpa's right leg is being restrained.

On the video recordings, Mr. Timpa is heard requesting "help" on 43 separate instances – 40 of the requests for "help" are following the onset of restraint by the police officers.

The cumulative times of compression of Mr. Timpa's torso by Officer Dillard's extremities are the following:

Officer Dillard's left knee compressed Mr. Timpa's torso for 14 min 4 sec (from 01.29 to 15.36, minus 3 sec).

Officer Dillard's right knee compressed Mr. Timpa's torso for 1 min 13 sec (from 02.14 to 3.27).

Officer Dillard's left-hand compressed Mr. Timpa's torso for 10 min 59 sec ([from 01.28 to 05.27, minus 1 sec] + [from 08.08 to 08:33] + [from 08:38 to 15:14]).

Officer Dillard's right-hand compressed Mr. Timpa's torso for 7 min 57 sec ([from 02.26 to 02:43] + [from 02.53 to 08.27] + [from 13:08 to 15.14]).

On video camera recordings, the toe of Officer Dillard's left boot is raised from the ground and can be seen to swing freely, signifying that Officer Dillard was exerting the total weight of his left lower extremity on Mr. Timpa's torso.

Vasquez Body Camera 04:17

Screenshot from Officer Vazquez's body camera at 04:17 (more than 2½ min after compression of Mr. Timpa's torso began). Officer Dillard's left knee is pressing down on Mr. Timpa's prone torso, with the toe of his left boot raised and can be seen to swing freely on video. Officer Dillard's left upper extremity, with elbow in extension, and left hand is compressing Mr. Timpa's torso.

The cumulative times of compression of Mr. Timpa's torso by Officer Vazquez's extremities are the following:

Officer Vazquez's left knee compressed Mr. Timpa's torso for 2 min 36 sec (from 01.29 to 04.05).

Officer Vazquez's left- or right-hand compressed Mr. Timpa's torso for 5 min 40 sec (from 09.28 to 15.08).

Dillard Body Camera 01:39

Screenshot from Officer Dillard's body camera at 01:39 (~02:17 on Officer Vazquez's clock). Officer Vasquez's left knee is pressing down on the left mid portion of Mr. Timpa's back. Officer Dillard's left hand is grasping and compressing the base of Mr. Timpa's neck.

EMS transported Mr. Timpa to Parkland Memorial Hospital, arriving in the ER at 11:12 PM. Dr. Alex Cohen pronounced Mr. Timpa dead at 11:30 PM.

Compression asphyxia

Based on my analysis of the events and my knowledge of human physiology and clinical medicine, I conclude that the cause of Mr. Timpa's death was a low level of oxygen in the cells of the body (asphyxia) caused by interference with breathing (compressive asphyxia). I base my conclusion on knowledge gained through undertaking research on pulmonary physiology for more than 40 years, practicing clinical medicine for more than 47 years, and education, knowledge and experience I gained over this time.

Three terms are used to describe the compromise of ventilation resulting from external force: compression asphyxia, traumatic asphyxia and crush asphyxia. Compression asphyxia is the term

that best fits the physiological processes involved *(Nolan-EMJ-2020; Rutty-Madea-2021)*. An external compressive force interferes with action of the diaphragm in expanding the rib cage leading to a decrease in tidal volume and alveolar ventilation *(Tobin-Clinics-Chest-Med-1988)*. The external force produces a decrease in end-expiratory lung volume (EELV), which decreases the stores of oxygen in the body *(Cherniack-PR-1970; Cross-Robin-JCI-1968)*. External compression produces an increase in stiffness of the lung and chest wall, which in turn leads to an increase in elastic work of breathing *(Laghi-Tobin-SOA-2003)*. External force compressing the torso also interferes with venous return, leading to a decrease in cardiac output.

Compressive asphyxia is also the cause of death in people who are involved in a crowd crush, such as at music concerts (ten deaths at the Houston Astroworld festival on November 7, 2021) or in sports stadiums. One of the most dramatic and best-studied examples was the Hillsborough disaster, which happened in a soccer stadium in Sheffield, England in 1989 *(Nolan-EMJ-2020; Rutty-Madea-2021)*. Altogether, 96 people died from compressive asphyxia in Hillsborough.

Hillsborough disaster 1989 (Sheffield, England): A few minutes after this photograph was taken, several of the people seen here were dead because of compressive asphyxia. There is no interference with the mouths of the people, but they are not able to expand their chest. As a result, they were not able to get oxygen into their body and they died of compressive asphyxia.

A series of physiological mechanisms resulted in a lethally low level of oxygen in Mr. Timpa's arterial blood, which made death inevitable. I describe the physiological processes individually. In reality, events were taking place simultaneously rather than sequentially. Before I discuss the abnormal physiological mechanisms that caused Mr. Timpa's death, I describe the physiology of normal breathing.

A. **Normal breathing**
Breathing is the process of bringing air from the atmosphere into the lungs in order to bring oxygen into the body and flush carbon dioxide out of the body.

Inspiration is an active process whereby contraction of the diaphragm (a sheet of muscle located between the chest and abdomen) and rib-cage muscles (muscles located between the ribs) causes pressure within the chest cavity to become more negative relative to the outside atmospheric pressure. The diaphragm is the most important muscle of inspiration *(Laghi-Tobin-SOA-2003; Sant'Ambrogio-1973)* despite its small mass (the normal diaphragm weighs 463.7 g (1.022 lb) *(Arora-Rochester-JAP-1982).* Diaphragmatic weight related to body weight according to the formula: 4.18 x body weight (kg) - 21.8 *(Arora-Rochester-JAP-1982).* Thus, Mr. Timpa's diaphragm weighed less than 1 lb (0.884 lb or 401.0 g): (4.18 x 101.15 kg) - 21.8 = 401.0 g (or 0.884 lb).

The pressure difference between the atmosphere and the interior of the chest cavity allows the air to move into the lungs passively along a pressure gradient. The amount of air inhaled with each breath is called the tidal volume. Expiration is a passive process whereby the elasticity of the tissues that constitute the chest wall and lung causes the chest cavity to deflate during exhalation and air to move from inside the chest cavity to the outside atmosphere *(Laghi-Tobin-SOA-2003)*.

Contraction of muscles of respiration causes expansion of the chest cavity in three directions. Contraction of the diaphragm causes the diaphragm to move downwards (in the direction of the toes) producing an increase of the space of the thoracic cavity for the lungs to expand *(DeTroyer-JAP-2016)*. Contraction of the diaphragm and intercostal muscles (muscles connecting one rib to the next rib) causes side-to-side expansion of the chest cavity, so called bucket-handle movement, and also front-to-back expansion of the chest cavity, so called pump-handle movement *(Laghi-Tobin-SOA-2003)*.

An especially important volume is end-expiratory lung volume (EELV). EELV is the amount of air that remains in the lung at the end of a normal breath. EELV plays a crucial role in storage of oxygen reserves *(Cherniack-PR-1970; Cross-Robin-1968)*.

B. **Physiologic impact of compressive force on respiratory and cardiovascular systems**
Compression of Mr. Timpa's torso, shoulders and legs in the prone position had several adverse effects on the respiratory and cardiovascular systems: (1) impaired expansion of the chest cavity in response to contraction of the diaphragm and rib-cage muscles; (2) inability to rotate the trunk to improve ventilation; (3) decrease in EELV and oxygen stores; and (4) decrease in cardiac output.

B. 1. <u>Impaired expansion of the chest cavity</u>
B. 1. a. *Ineffective diaphragmatic contraction*
While Mr. Timpa was prone, Officer Dillard's left knee, left-hand, right-hand, and, at times, right knee, and, also, Officer Vazquez's left knee, left-hand and right-hand compressed Mr. Timpa's torso against the ground. Compression of the torso and the overweight abdomen against the ground increased increased the pressure within Mr. Timpa's abdominal cavity, which, in turn, impeded the downward movement of the diaphragm. Consequently, Mr. Timpa's diaphragm was not able

to generate adequate tidal volumes. During resting breathing, average tidal volume is 383±85 (SD) ml (range, 211-575 ml) *(Tobin-1983)*. About 75-90% of tidal volume is achieved by contraction of the diaphragm, and the remaining portion results from contraction of rib-cage muscles *(Sant'Ambrogio-1973)*.

Dillard Body Camera 03:57

Screenshot from Officer Dillard's body camera at 03:57 (~04:35 on Officer Vazquez's clock). Officer Dillard's left upper extremity, with elbow in extension, is compressing Mr. Timpa's left shoulder. Tension visible on the dorsum of Officer Dillard's left hand signifies use of firm force when pressing down on Mr. Timpa's left shoulder.

B. 1. b. *Ineffective rib-cage expansion*

During the restraint of Mr. Timpa, the front portion of his chest (the region surrounding his breastbone) was pushed against the ground by Officer Dillard's left knee, left-hand, right-hand, and, at times, right knee, and, also, by Officer Vazquez's left knee, left-hand and right-hand. Consequently, Mr. Timpa's diaphragm and rib-cage muscles were not able to achieve normal front-to-back expansion (pump-handle movement) of his chest cavity and, thus, the size of his tidal volume was diminished *(Tobin-Clinics-Chest-Med-1988)*.

Vasquez Body Camera 09:29

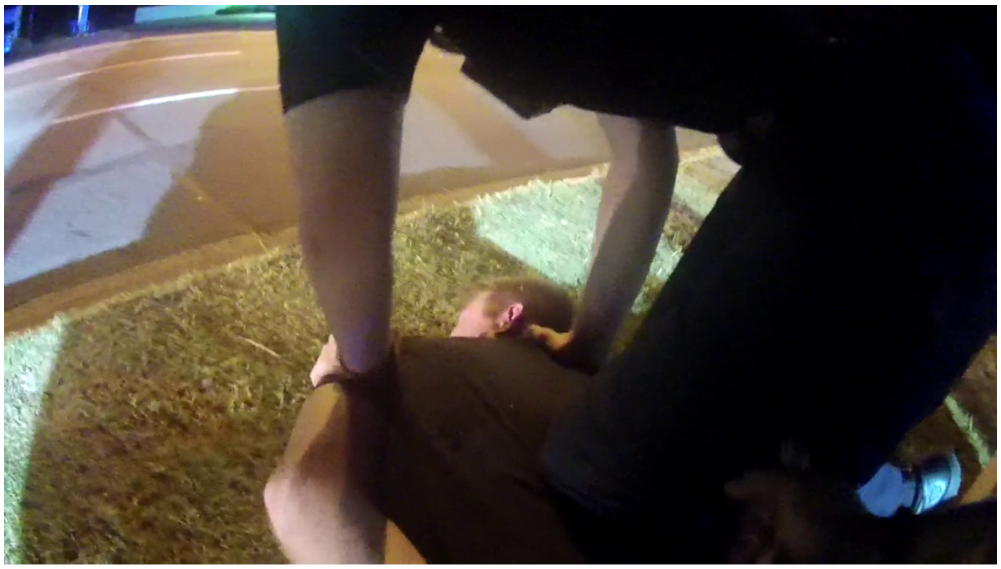
Screenshot from Officer Vazquez's body camera at 09:29 (~ 8 min after compression of Mr. Timpa's prone torso began). On the bottom left of the frame, Officer Vasquez's left hand is pressing down on Mr. Timpa's left-lower back. Officer Dillard's left knee is pressing down on Mr. Timpa's prone torso, with toe of left boot raised and can be seen to swing freely on video. The orientation of Officer Dillard's left thigh is erect. Officer Dillard's left upper extremity, with elbow in extension, and left hand is compressing Mr. Timpa's left shoulder.

In summary, the compressive forces pushing down on Mr. Timpa's rib cage and abdomen against the ground hindered the ability of his diaphragm and rib-cage muscles to expand his chest cavity (pump-handle action and downward movement of the diaphragm), thereby causing alveolar hypoventilation and decreasing the amount of air entering his lungs (smaller tidal volume) *(Hussain-Pardy-1985)*. When tidal volume falls below 150 ml, it ventilates only dead space and air does not reach the site (the alveoli) at which oxygen is delivered to the bloodstream and carbon dioxide is removed. The low tidal volume resulted in less oxygen being brought into the air sacs (alveoli) of the lung and its transfer into the blood stream, thereby causing the oxygen level in the body to fall to a lethal level (asphyxia).

B. 2. Inability to rotate trunk to improve ventilation

Because of the compressive forces acting on Mr. Timpa's torso throughout the time that he was restrained prone on the ground, Mr. Timpa's only means to generate a breath (generate tidal volume) was to rotate his body on to either his left- or right-side (left- or right-lateral decubitus position). Rotating his trunk and lifting one or other side of his chest off the ground would have helped with front-back expansion (pump-handle movement) of the chest, improving alveolar ventilation.

Appendix #2 lists several occasions where Officers refer to Mr. Timpa as "squirming" (03:02, 06:02, 06:09, 13:00, 13:44). In the depositions of Officers Dillard, Vasquez and Dominguez, the police officers repeatedly refer to their efforts to prevent Mr. Timpa from squirming and trashing (Officer Dillard deposition: pages 124, 126, 157, and 221; Corporal Dominguez deposition: pages 42, 122, 229, 295, and 296; Officer Vasquez deposition: pages 115, 117, 123, 124, and 262. On page 124 of his deposition, Officer Vasquez describes squirming as meaning to "move left, move right …enough to move me and other officers." The Police Officers failed to recognize that Mr. Timpa's struggling movements were his attempts to unseat the compressive forces bearing down on his chest in order to restore alveolar ventilation.



Vasquez Body Camera 04:56

Screenshot from Officer Vazquez's body camera at 04:56 (~3½ min after compression of Mr. Timpa's prone torso began). Officer Dillard's left knee is pressing down on the mid to lower portion of Mr. Timpa's back, with toe of left boot bobbing up and down on video. Officer Dillard's left hand (with left elbow extended) is grasping and pushing down on Mr. Timpa's left shoulder. Officer Dillard's right hand (with right elbow extended) is grasping and pushing down on the base of Mr. Timpa's neck.

By grasping Mr. Timpa's shoulders and forcing them down to the ground, by kneeling on Mr. Timpa's torso, and by manually pushing down on Mr. Timpa's torso, Officer Dillard and Officer Vasquez prevented Mr. Timpa from rotating his body to a lateral decubitus position in order to restore alveolar ventilation. Corporal Dominguez and Officer Rivera retrained Mr. Timpa's legs, which impaired Mr. Timpa's ability to rotate his pelvis and, thus, move his spine and trunk to a lateral decubitus position and restore alveolar ventilation.

Rivera Body Camera 03:48

Screenshot from Officer Rivera's body camera at 03:48 (~06:12 on Officer Vazquez's clock). Officer Rivera is using two hands to restrain Mr. Timpa's left foot. Corporal Dominguez is visible in the upper right corner, reaching under the bench.

B. 3. Decrease in end-expiratory lung volume (EELV)

B. 3. a. *Factors contributing to a decrease in EELV*

B. 3. a. i. *Prone.* Moving a person from upright posture to prone position produces a 23.7% decrease in EELV *(Moreno-Lyons-JAP-1961)*. Based on height, age and sex, Mr. Timpa's EELV is calculated as 3,410 ml in the sitting posture *(Quanjer-1993).* (EELV (ml) = 1000* ((2.34*Height[m]) + (0.009*Age[y]) - 1.09) = 1000*((2.34 * 1.8) + (0.009 * 32) - 1.09) = 3410 ml). Turning Mr. Timpa prone decreased his EELV from 3,410 ml to 2,601.8 ml, representing an absolute decrease in EELV of 808.2 ml (3410*0.237 = 808.2 ml).

B. 3. a. ii. *Application of compressive force on chest and abd*omen. Application of compressive force produces an additional decrease in EELV. Applying a very small positive pressure (+9.4±1 (SE) cm H_2O) on the ventral (anterior) thorax and abdomen is sufficient to produce 732±74 ml decrease in EELV *(Heinzer-White-AJRCCM-2005)*.

Employing the most widely used formula to estimate body surface area (BSA)—BSA= 0.016667 × $W^{0.5}$ × $H^{0.5}$ *(Mosteller-NEJM-1987)*—Mr. Timpa's BSA was 2.25 m^2 (0.016667 × $101.15^{0.5}$ × $180.3^{0.5}$). According to the "rule of 9" *(Wallace-Lancet-1951)*, surface area of the dorsal (back) portion of the thorax is 9% of total BSA and surface area of the lumbar portion is also 9%. Mr. Timpa's dorsal and lumbar surface area was 4,052 cm^2 (2.25 * (0.09 + 0.09) = 0.4052 m^2 = 4,052 cm^2).

While Mr. Timpa was being restrained prone on the ground, Officer Dillard's left lower extremity (with toe raised and swinging freely) together with his left-hand and right-hand were pressing down on Mr. Timpa's back producing a gravitational force of 24.326 kg. This gravitational force, 24.326 kg, was calculated as follows. Officer Dillard's body weight was 72.6 kg (160 lb), police-gear weight 30 lb (13.6 kg; as usually reported), resulting in a total weight of 86.2 kg (190.04 lb). The weight of Officer Dillard's left lower extremity was 16.68% of his total weight, weight of his right arm was 5.77% of his total weight, and weight of his left arm was 5.77% of his total weight *(Plagenhoef-RQES-1983)*—that is, 14.378 kg (86.2*0.1668 = 14.378) plus 4.974 kg (86.2*0.0577 = 4.974) plus 4.974 kg (86.2*0.0577 = 4.974), resulting in a combined weight of 24.326 kg (14.378 + 4.974 +4.974 = 24.326). The resulting compressive force decreased Mr. Timpa's EELV by 467 ml (19.2 ml/kg * 24.326 = 467 ml) from unencumbered position. Consequently, the decrease in EELV caused by the combination of placing Mr. Timpa prone (808 ml) plus the effect of Officer Dillard's left knee, left-hand and right-hand (467 ml) was 1275 ml (808+467=1275) in absolute units or 37.4% (1275/3410=37.4%) expressed as a percentage.

In addition to the compression resulting from Officer Dillard's left knee, left-hand, and right-hand (I have omitted the compression resulting from Officer Dillard's right knee), further compression resulted from Officer Vazquez's left knee pressing down on Mr. Timpa's back. Because I was not able to get a clear view as to whether (or not) Officer Vasquez's left toe was raised from the ground, my calculation of gravitational force omits the effect of his foot. The gravitational force of Officer Vazquez's left knee was 14.87 kg. This gravitational force, 14.87 kg, was calculated as follows. Officer Vazquez's body weight was 83.9 kg (185 lb), police-gear weight 30 lb (13.6 kg), resulting

in a total weight of 97.5 kg (215 lb). The weight of Officer Vazquez's left lower extremity (minus the foot) was 15.25% of his total weight *(Plagenhoef-RQES-1983)*—that is, 14.87 kg (97.5.*0.1525=14.87). The resulting compressive force decreased Mr. Timpa's EELV by 286 ml (19.2 ml/kg * 14.87 kg= 286 ml). Later in the restraint, Officer Vasquez's used his left-hand alternating with his right-hand to compress Mr. Timpa's torso; I have omitted these actions from the calculation of the gravitational force exerted by Officer Vasquez.

The combined gravitational force of Officer Dillard and Officer Vazquez, 39.19 kg (24.326+14.87=39.19 kg), produced a cumulative decrease of 753 ml (467+286=753 ml) in Mr. Timpa's EELV from the unencumbered prone position. Consequently, the combination of placing Mr. Timpa prone (808 ml) plus the combined gravitational forces of Officer Dillard and Officer Vazquez caused a decrease in EELV of 1,561 ml (808 ml + 753 ml = 1561 ml) in absolute units or 45.8% (1561/3410=45.8%) expressed as a percentage.

B. 3. b. Consequences of a decrease in EELV

B. 3. b. i. *Increase in effort to breathe*

Respiratory work. To achieve a given tidal breath (normal breathing), the respiratory muscles must work to overcome the resistive forces of the airways and the elastic forces developed by the lung and chest wall. In the presence of increased airway resistance (airway narrowing) or decreased elastic forces (stiff lungs), increased pressure is required to achieve a given tidal volume with the consequent increase in work of breathing *(Tobin-HP-2012)*.

The amount of effort (force) used by the respiratory muscles to expand the lungs and chest wall, or work of breathing, can be quantified as pressure generated by the respiratory muscles over the time of contraction, known as pressure-time product (PTP). Normal PTP is 94 ± 12 (SE) cm H_2O/s/min *(Jubran-Tobin-1997)*. Of the total PTP expended by the respiratory muscles, 53.6 cm H_2O/s/min (57%) is used to overcome the elastic (stiffness) forces of the lung and the chest wall (elastic work); and the remainder of PTP (40.4 cm H_2O/s/min) is used to overcome airway resistance (resistive work).

Elastic work. In a 32-year-old man, average stiffness of the lungs (pressure needed to expand lungs) is 3.55 cm H_2O/l, and average stiffness of the chest wall is 3.47 cm H_2O/l, resulting in total stiffness of 7.58 cm H_2O/l *(Mittman-1965)*. When breathing freely, the portion of Mr. Timpa's PTP not related to airway narrowing was equally divided between effort to expand the lungs (26.8 cm H_2O/s/min) and the chest wall (26.8 cm H_2O/s/min).

Lung stiffness. For 1% decrease in EELV, lung stiffness increases by 1.35% *(Eberlein-2014)*. Officer Dillard's compression of Mr. Timpa's torso caused 1,275.2 ml decrease in EELV (equivalent to 37.4% decrease in EELV) and 50.5% increase in lung stiffness (37.4% [decrease in EELV] * 1.35 = 50.5%) from 3.55 cm H_2O/l to 5.3 cm H_2O/l. Torso compression accordingly increased Mr. Timpa's PTP to expand the lung by 50.5% over the unencumbered state: 40.3 vs. 26.8 cm H_2O/s/min.

During the restraint, Officer Vasquez's compression of Mr. Timpa's torso caused 1,094 ml decrease in EELV (equivalent to 32.1% decrease in EELV) and 43.3% increase in lung stiffness (32.1% [decrease in EELV] * 1.35 = 43.3%) from 3.55 cm H_2O/l to 5.1 cm H_2O/l. Torso

compression accordingly increased Mr. Timpa's PTP to expand the lung by 43.3% over the unencumbered state: 38.4 vs. 26.8 cm H_2O/s/min.

In summary, the combined compressive forces of Officer Dillard and Officer Vasquez on Mr. Timpa's back caused the PTP to overcome the elastic forces of the lungs to increase from 26.8 cm H_2O/s/min (during unencumbered sitting) to 78.7 cm H_2O/s/min during the restraint.

Chest wall stiffness. For 1 kg increase in chest compression, chest-wall stiffness increases by 0.23 cm H_2O/l *(Sharp-1964)*. Officer Dillard's compressing Mr. Timpa's torso increased the PTP to expand the chest wall of Mr. Timpa by 161% over the unencumbered state: 70.0 vs. 26.8 cm H_2O/s/min.

During the restraint, Officer Vasquez's compressing Mr. Timpa's torso increased the PTP to expand the chest wall of Mr. Timpa by 98.5% over the unencumbered state: 53.2 vs. 26.8 cm H_2O/s/min.

In summary, combined compressive forces of Officer Dillard and Officer Vasquez on Mr. Timpa's torso caused the PTP to overcome the elastic forces of the chest wall to increase from 26.8 cm H_2O/s/min (during unencumbered sitting) to 123.2 cm H_2O/s/min during the restraint.

Total respiratory work. Mr. Timpa's total PTP is the sum of elastic work (201.95 cm H_2O/s/min) and resistive work (40.4 cm H_2O/s/min). Accordingly, in the restrained prone position, Mr Timpa's total PTP increased from 94 (unencumbered sitting) to 242.4 cm H_2O/s/min (258% increase from unencumbered sitting).

The 2.58-fold increase in respiratory work imposed by the prone position and compressive forces of Officer Dillard and Officer Vasquez produced a huge burden on Mr. Timpa's diaphragm and rib-cage muscles whose function was already severely compromised. As time passed, the imbalance between the increase in mechanical load and reduced pressure-generating capacity of the respiratory muscles worsened, causing the muscles to go into a state of impaired excitation-contraction coupling of the actin and myosin molecules inside the filaments of the respiratory muscles, resulting in a decrease in muscle force *(Laghi-Tobin-SOA-2003)*. The decrease in muscle force then caused tidal volume to decrease to such an extent that it was no longer bringing enough air to the lung alveoli (air sacs), leading to decreased level of oxygen in blood (asphyxia).

B. 3. b. ii. *Decrease in oxygen stores*

The reservoir of oxygen in the body is small, and stores of oxygen retard the rate of change in arterial oxygen tension (PaO_2) so that values are maintained within narrow limits *(Cherniack-PR-1970; Cross-Robin-JCI-1968)*. Oxygen is stored mainly in the lung (as a gas in EELV) and in blood. Normally, oxygen stores are being constantly replenished by ventilation.

The oxygen stored in the lung is the product of the fraction of alveolar oxygen (F_AO_2) at end-expiration (0.155) and EELV *(Nunn-1987)*. In the unencumbered sitting position, oxygen stores in the lungs of Mr. Timpa were 528.55 ml (F_AO_2* EELV= 0.155*3410 ml=528.55 ml). The amount of oxygen stored in Mr. Timpa's blood (1.34 [ml/gm] * hemoglobin [gm/dl] * arterial oxygen saturation * blood volume [dl]) was 1,184 ml (1.34 ml/gm *15 gm/dl * 0.98 * 60.11 dl =1184 ml) (blood volume was estimated based on Mr. Timpa's height, weight and sex). The

amount of oxygen dissolved in plasma (PaO_2*solubility of oxygen *blood volume) was 17.74 ml (95.2*0.0031* 60.11 = 17.74 ml). Accordingly, Mr. Timpa's total oxygen stores in the unencumbered sitting position were 1,730 ml (528.55+1184+17.74=1730 ml).

Decrease in EELV. A decrease in EELV causes a necessary decrease in the amount of oxygen that is stored in the lungs. As a result of being prone and the combined compressive force of Officer Dillard's left lower extremity, left-hand and right-hand, and Officer Vazquez's left knee, oxygen stores in the lung decreased from 528.55 ml during unencumbered sitting to 286.6 ml (0.155*1849=286.6 ml) during combined compressive actions of Officer Dillard and Officer.

Decrease in replenishment of oxygen stores. Normally, oxygen stores are being constantly replenished by ventilation. Mr. Timpa's alveolar ventilation, however, was markedly decreased as a result of placement in the prone position and compression of his torso against the ground. The decrease in alveolar ventilation impaired the replenishment of already-compromised oxygen stores leading to further decrease in arterial oxygen (asphyxia).

Increase in oxygen consumption. In the unencumbered sitting position, approximately two-thirds (1,153 ml) of Mr. Timpa's total body oxygen stores (1,730 ml) were rapidly exchangeable *(Cross-Robin-JCI-1968)*. Given that resting oxygen consumption is normally 250 ml/min, complete exhaustion of Mr. Timpa's oxygen stores would have occurred in 4.61 minutes (4 min 37 sec) after total cessation of ventilation (apnea) *(Cross-Robin-JCI-1968)*.

Because of the struggle to compensate for the respiratory challenges imposed upon him during the restraint, Mr. Timpa's oxygen consumption was substantially higher than at rest, as reflected by the up to 2.58-fold increase in PTP. Moreover, the amount of oxygen stored in his lungs was substantially reduced as a result of the decrease in EELV. Accordingly, the increase in oxygen consumption during the restraint depleted his oxygen stores more quickly than at rest. For example, if Mr. Timpa's oxygen consumption doubled from normal (500 ml/min) during the restraint (a conservative assumption), and using the calculated total oxygen stores during the restraint of 1,488 ml (287 [lungs] +1184 [blood] +17.7 [dissolved in plasma] = 1488 ml), of which 992 ml (0.667* 1488=992 ml) were exchangeable, it would have taken 1 min 59 sec (992/500=1.98 min) for Mr. Timpa to have completely used up his oxygen stores after cessation of breathing, as contrasted with 3 min 58 sec (992/250=3.97) at normal oxygen consumption.

In reality, Mr. Timpa's oxygen consumption is estimated to have increased by 2.58 fold from normal (645 ml/min) during the restraint, and using the calculated total oxygen stores during the restraint of 1,488 ml (287 [lungs] +1184 [blood] +17.7 [dissolved in plasma] = 1488 ml), of which 992 ml (0.667*1488=992 ml) were exchangeable, it would have taken 1 min 32 sec (992/645=1.54 min) for Mr. Timpa to have completely used up his oxygen stores after cessation of breathing, as contrasted with 3 min 58 sec (992.5/250=3.97 min) at normal oxygen consumption.

B. 4. Impairment of cardiovascular performance

To cope with the many physiological challenges Mr. Timpa was facing, it was imperative that his heart would be able to transport sufficient oxygen to body organs and muscle groups *(Jubran-Tobin-1998)*. On the contrary, Mr. Timpa's cardiovascular performance was decreased in a predictable manner as a result of being turned prone and the compression exerted by Officer

Dillard and Officer Vasquez on his torso. Based on research into injuries occurring in crowd-crush incidents (such as the Hillsborough disaster), it is known that forces applied to the chest and abdomen (even without affecting the mouth or neck) cause rapid death from compressive asphyxia and impaired cardiac performance *(Nolan-Rutty-EMJ-2021; Channer-BMJ-1989)*.

Severe compression of the torso causes an increase in intrathoracic pressure, which compresses the inferior vena cava, the large vein within the abdominal cavity that carries deoxygenated blood from the lower body to the heart. Compression of the vena cava decreases venous return to the heart, and, in turn, reduces cardiac output. In healthy subjects, chest strapping (corset), associated with a decrease in EELV from 4,080 to 2,078 ml, caused 16.09% decrease in cardiac output *(Klineberg-Hyatt-JAP-1981)*. When surgery is performed on the spine, patients are turned prone and cardiac output inevitably decreases by more than 20%, primarily as a result of decrease in stroke volume (blood pumped per heartbeat) *(Edgcombe-BJA-2008; Sudheer-2006; Yokoyama-1991; Hatada-1991)*.

Mr. Timpa's cardiac output was further decreased by the increase in pressure within the abdominal cavity consequent to the compressive force exerted by Officer Dillard and Officer Vasquez on Mr. Timpa's torso and obese abdomen against the ground, and the automatic recruitment of expiratory muscles caused by an increase in elastic load *(Laghi-Tobin-JAP-2020; Parthasarathy-Tobin-JAP-2007)*. When pressure within abdomen is increased by 40 mmHg, cardiac output falls by 17% *(Kashtan-1981)*. The increase in abdominal pressure compresses the vena cava, decreasing return of blood to heart *(Thompson-1989)*.

The decrease in Mr. Timpa's cardiac output as a result of multiple factors meant that his heart was not able to pump sufficient blood and oxygen to the large muscles of his torso, which he was forcefully contracting in an attempt to rotate his trunk in order to free and expand his rib cage, enabling it to engage in the act of breathing. Decreased cardiac output also decreased blood and oxygen supply to the diaphragm and intercostal muscles, which he was forcefully contracting in an attempt to expand his rib cage. With the heart unable to supply sufficient oxygen, excitation-contraction coupling of these muscle groups became drastically impaired, and terminal failure of muscle contraction meant that breath size (tidal volume) became so small that a very small amount of oxygen was available in the lung alveoli (air sacs) for gas exchange *(Tobin-HP-2012;Viires-1983)*. The small amount of oxygen in the alveoli compounded by diminished cardiac output and blood flow caused blood oxygen to fall below the level necessary to maintain consciousness and prevent cardiac arrest.

B. 5. Alveolar hypoventilation

The ultimate consequence of compressive asphyxia is a decrease in alveolar ventilation, which axiomatically produces an increase in arterial $PaCO_2$ *(Tobin-HP-2012)*. The physiological mechanisms leading to alveolar hypoventilation are best understood by examination of one of the canonical equations of pulmonary physiology, namely the alveolar ventilation equation *(Tobin-HP-2012)*, which dictates that alveolar CO_2 tension ($PACO_2$), and thus arterial $PaCO_2$, is determined by the ratio of the body's metabolic production of CO_2 (VCO_2) divided by alveolar ventilation (V_A):

$$PACO_2 = K * VCO_2 / V_A$$

If VCO_2 is expressed as ml/min, in STPD (standard temperature, pressure and dry) conditions, and V_A as L/min, in BTPS (body temperature, ambient pressure and gas saturated with water vapor) conditions, then *K* has the dimension of a pressure and is equal to 0.86 *(Otis-HP-1964)*. This relationship necessitates that at a given VCO_2, any decrease in V_A must be accompanied by a reciprocal increase in arterial $PaCO_2$. Because arterial $PaCO_2$ is in equilibrium with alveolar $PACO_2$ and because V_A equals minute ventilation (V_E) minus dead-space (V_D/V_T) ventilation, the alveolar ventilation equation can be rearranged as:

$$PaCO_2 = K * VCO_2 / V_E (1 - V_D/V_T)$$

Minute ventilation (V_E) is the product of tidal volume (V_T) and respiratory frequency (f). Because significant change in respiratory frequency (rate) was not observed and there is no reason to believe that Mr. Timpa's dead space was abnormal during the restraint, his arterial $PaCO_2$ was determined solely by levels of VCO_2 and tidal volume (V_T).

During the restraint, Mr. Timpa's chest expansion was impaired consequent to (a) being placed in the prone position, (b) decreased expansion of the chest cavity consequent to downward pressure on his torso against the ground exerted by Officer Dillard's left knee, left-hand, right-hand, and, at times, right knee, and, also, Officer Vazquez's left knee, left-hand and right-hand, and (c) increased stiffness of the chest wall and (d) increased stiffness of lung tissue as reflected by increases in chest-wall pressure-time product (PTP) and lung PTP. All of these physiological factors conjoined to produce a decrease in Mr. Timpa's tidal volume.

While always assiduously emphasizing that restraint in the prone position with weights placed on the back do not result in "clinically significant" decreases in pulse oximetry recordings of oxygen saturation, Dr. Chan and his group have reported significant decreases in lung volumes in the restraint setting:

- 20.78% decrease in maximum voluntary ventilation (MVV) with hobble restraint *(Chan-Clausen-AEM-1997)*,
- 24.47% decrease in forced expired volume in one second (FEV1) for prone maximal restraint position (PRMP, also known as hogtie or hobble) with 50 lb weight placed on the back *(Chan-Clausen-AJFMP-2004)*,
- 30.3% decrease in MVV for prone maximal restraint position (PRMP), with 102.3 kg (225.53 lb) of weight on the back *(Michalewicz-Chan-JFS-2007)*; the 95% confidence interval of 54% for MVV in the Michalewicz-Chan experiment signifies that some of their subjects experienced 44% decrease in MVV

A 35.0% decrease in FEV1 in subjects restrained in the prone position with 75 kg (165.35 lb) of sand-filled bags placed evenly across the back was reported by physiologists at Imperial College, London *(Cary-J-Physiol-2000)*.

From the above, it is evident that restraining subjects in the prone position produces a decrease in lung volume that ranges between 20.78% and 35.0% decrements.

Using the framework of normal blood gases and breathing pattern in a 32-year-old man, I calculate the changes in lung volumes and gas exchange that Mr. Timpa experienced during the restraint. The expected breathing pattern in a 32-year-old man is tidal volume (V_T), 383 ml, respiratory frequency (f), 16.7 breaths/min, and minute ventilation (V_E), 6.396 L/min *(Tobin-Chest-1983)*.

Metabolic production of CO_2 (VCO_2) at rest is typically 200 ml/min *(Tobin-HP-2012)*; V_D/V_T is typically 0.40, yielding an anatomical dead space of 153.2 ml (383*0.40=153.2 ml).

Mr. Timpa's resting arterial $PaCO_2$ is calculable according to the (rearranged) alveolar ventilation equation, $K * VCO_2 / V_E (1 - V_D/V_T)$, as follows: $PaCO_2 = [0.86*200] / [6.396* (1 - (153.2/383))]$, thus 172 / (6.396*0.6), yielding an arterial $PaCO_2$ of 44.82 mmHg.

The expected alveolar-to-arterial oxygen (PAO_2-PaO_2) difference in a 32-year-old man is 3.3 mmHg *(Crapo-AJRCCM-1999)*. Arterial PaO_2 is calculable according to the formula: $PaO_2 = PAO_2$ - 3.3 mmHg, where PAO_2 is oxygen tension inside the alveoli and PaO_2 is oxygen tension in arterial blood. PAO_2 is calculable using another canonical equation of pulmonary physiology, the alveolar gas equation:

$$PAO_2 = F_IO_2 * (P_B - P_{H2O}) – (PaCO_2 / RQ)$$

where F_IO_2 = fractional inspired oxygen concentration (0.21 while breathing room air), P_B = barometric pressure (760 mmHg at sea level), P_{H2O} = water vapor pressure (47 mmHg at 37°C) and RQ = respiratory exchange ratio, usually taken as 0.80. Expected PAO_2 with an arterial $PaCO_2$ of 44.82 mmHg is 94 mmHg (=0.21*(760-47) - (44.82/0.8) = 149.7 - 56.02=93.7). Thus, arterial PaO2 is 91 mmHg (94 - 3.3 = 90.7).

If Mr. Timpa's tidal volume decreased by 20.78% (the lower decrement in lung volume reported by Dr. Chan *(Chan-Clausen-AEM-1997)*) consequent to the compressive restraint exerted through the actions of Officer Dillard and Officer Vazquez, the resultant tidal volume (V_T) would be 303.4 ml and minute ventilation (V_E) 5.07 L/min. Employing the (rearranged) alveolar ventilation equation, arterial $PaCO_2$ that results with a 20.78% decrease in volume (tidal volume (V_T) and minute ventilation (V_E)) is calculable as 68.5 mmHg ($PaCO_2$=0.86*200/5.07*[1 - (153.2/303.4)] = 172/2.51 = 68.5 mmHg). Again, using the alveolar gas equation, PAO_2 at an arterial $PaCO_2$ of 68.5 mmHg is calculable as 64 mmHg (=0.21*(760-47)- (68.5/0.8) =149.7-85.6=64). Thus, arterial PaO_2 at arterial $PaCO_2$ 68.5 mmHg is calculable as 60.7 mmHg (=64-3.3=60.7).

If Mr. Timpa's tidal volume decreased by 35.0% (the upper decrement in lung volume reported in restraint experiments *(Cary-J-Physiol-2000; Michalewicz-Chan-JFS-2007)*) consequent to the compressive restraint exerted by Officer Dillard and Officer Vazquez, the resulting tidal volume will be 248.5 ml and minute ventilation 4.15 L/min. Employing the (rearranged) alveolar ventilation equation, arterial $PaCO_2$ that results with a 35.0% decrease in volume (tidal volume (V_T) and minute ventilation (V_E)) is calculable as 108.1 mmHg ($PaCO_2$=0.86*200/4.15*[1 - (153.2/248.5)] = 172/1.591=108.1 mmHg). Again, using the alveolar gas equation, PAO_2 at an arterial $PaCO_2$ of 108.1 mmHg is calculable as 14.6 mmHg (=0.21*(760-47) - (108.1/0.8) =149.7-135.1=14.6). Thus, arterial PaO_2 at arterial $PaCO_2$ 108.1 mmHg is 11.3 mmHg (=14.6-3.3=11.3).

The above calculations demonstrate that a 35.0% decrease in Mr. Timpa's tidal volume consequent to the compressive restraint would have produced an arterial PaO_2 far below 37 mmHg *(Nunn-1987-p474)*, the minimum PaO_2 that is compatible with consciousness and voluntary contraction of head and neck muscles. The arterial $PaCO_2$ that will achieve arterial PaO_2 37 mmHg is calculable from knowledge of alveolar PAO_2 (derived from the alveolar-to-arterial oxygen [PAO_2-PaO_2] difference) and then employing the alveolar gas equation to determine the requisite arterial

$PaCO_2$. For arterial PaO_2 37 mmHg, alveolar PAO_2 is 40.3 mmHg (37.0 + 3.3 = 40.3). By rearranging the alveolar gas equation, arterial $PaCO_2$ is calculable as $PaCO_2$=-(PAO_2-(0.21* (760-47))/0.80. Thus, arterial $PaCO_2$ at arterial PaO_2 37 mmHg is 87.5 mmHg ($PaCO_2$ =-(PAO_2 - 149.7)/0.80 = -(40.3-149.7)/0.8 = 87.52.).

If Mr. Timpa did not experience an increase in metabolic production of CO_2 (VCO_2) (which is not likely) and his dead space (V_D) did not increase (which is likely), then tidal volume (V_T) necessary to achieve an arterial $PaCO_2$ 87.5 mmHg can be calculated by a further re-arrangement of the alveolar ventilation equation:

$$V_T = K*VCO_2 / (PaCO_2 * \text{frequency}) + V_D$$

Taking VCO_2 200 ml/min and K as 860 (because V_T is expressed in ml), tidal volume (V_T) is calculable as 271.0 ml (=860 * 200) / (87.5*16.7) + 153.2 = (172,000 /1,461.25) + 153.2 = 117.71 + 153.2 = 270.9). The latter V_T is 70.73% of Mr. Timpa's baseline tidal volume (V_T) 383 ml (271.3 / 383 = 0.7073 = 70.73%). In other words, when Mr. Timpa reached arterial $PaCO_2$ 87.5 mmHg (and arterial PaO_2 37 mmHg *(Nunn-1987-p474))*, his V_T had decreased by 29.3% (1-0.7073=0.293) from his baseline V_T of 383 ml.

If Mr. Timpa experienced an increase in metabolic production of CO_2 (VCO_2), consequent to cocaine consumption and skeletal-muscle exertion as he struggled against superimposed officers during the restraint, he would have reached arterial $PaCO_2$ 87.5 mmHg (and arterial PaO_2 37 mmHg *(Nunn-1987-p474)* with smaller decrements in minute ventilation (V_E) and tidal volume (V_T) from baseline values. This point can be grasped by doing equivalent calculations for metabolic production of CO_2 (VCO_2) 250 ml/min rather than VCO_2 200 ml/min (which was used in the preceding paragraph). Employing the (rearranged) alveolar ventilation equation ($PaCO_2$ = $K * VCO_2 / V_E (1 - V_D/V_T)$, Mr. Timpa would have reached arterial $PaCO_2$ 87.5 mmHg (and arterial PaO_2 37 mmHg) at minute ventilation (V_E) of 5.02 l/min and tidal volume (V_T) 300 ml. That is, a 21.67% decrease in V_T from baseline (383 ml) will achieve $PaCO_2$ 87.5 mmHg when VCO_2 is 250 ml/min.

If Mr. Timpa's metabolic production of CO_2 (VCO_2) increased to 300 ml/min, he would have reached arterial $PaCO_2$ 87.5 mmHg (and arterial PaO_2 37 mmHg) at minute ventilation (V_E) 5.54 l/min and tidal volume (V_T) 332 ml. That is, a 13.31% decrease in V_T from baseline (383 ml) (383 - 332 = 51; 51/ 383 = 13.31%).

The preceding calculations make it clear that the decrements in lung volumes consequent to the compressive forces exerted by Officer Dillard and Officer Vazquez were more than sufficient to produce a decrease in alveolar ventilation that resulted in asphyxia. A decrease in arterial PaO_2 to 37 mmHg resulted from a 29.3% decrease in baseline tidal volume (V_T) (from 383 ml to 271 ml) when metabolic production of CO_2 (VCO_2) was normal, 200 ml/min. If, as more likely, metabolic production of CO_2 (VCO_2) was elevated, arterial PaO_2 37 mmHg resulted from a 21.67% decrease in baseline tidal volume (V_T) at VCO_2 of 250 ml/min or from a 13.31% decrease in baseline V_T at VCO_2 of 300 ml/min. The extent of these decrements in lung volume are well within the range of decreases in lung volume during restraint reported by Dr. Chan and his group *(Chan-Clausen-AEM-1997; Michalewicz-Chan-JFS-2007)*.

The striking difference between the experiments conducted by Dr. Chan and events leading to the death of Mr. Timpa is the duration of the restraint. As discussed later in my report, Dr. Barnard, Dr. Bird and Dr. Kroll claim that research studies conducted by Dr. Chan do not produce "clinically significant" decreases in pulse oximetry readings of oxygen saturation (the major problems with use of this tool for detecting changes in arterial oxygen tension in healthy subjects are discussed on **pages 32-34** of my report). When questioned about the Michalewicz-Chan experiment *(Michalewicz-Chan-AEM-1997)*, none of the deponents mentioned the glaring differences in the duration of the Michalewicz-Chan experiment as contrasted with the length of time Mr. Timpa was restrained. The restraint and struggle in the Michalewicz-Chan experiment was limited to 60 sec. Yet, two of the 30 subjects withdrew from the study "because they were psychologically unable to tolerate restraint…they were frightened of being restrained." Among the 27 subjects that did not withdraw, Michalewicz-Chan noted "The struggle was physically difficult for subjects. In spite of continued verbal encouragement, the intensity of movement was visibly waning in all subjects by the end of the 60-sec trial" *(Michalewicz-Chan-AEM-1997)*. Officer Dillard placed his left knee on Mr. Timpa's torso at 01:29 and it remained on the torso until 15:36 (except for 3 sec); at the time Mr. Timpa made his last voluntary muscle contraction (11:55), he had been restrained for more than 10 min 20 sec—more than 10 times longer than the duration of the controlled Michalewicz-Chan experiment where all subjects were visibly waning "by the end of the 60-sec trial."

The above quoted extracts from the Michalewicz-Chan article underscore the overarching conclusion of this section of my report. A 20-30% decrease in tidal volume (V_T) imposed for 10 minutes, and crucially with further imposed constraints to prevent a detainee from taking the vital countervailing steps to achieve hyperventilation (necessitating as much as a 6-fold increase in minute ventilation) and blow off CO_2 (when VCO_2 is elevated) in order to protect against fatal hypoxemia (as described on **pages 35-37** of my report) can be predicted mathematically to produce death from asphyxia. Yet, this vital protective strategy was not even contemplated in the articles by Dr. Chan and coworkers.

B. 6. <u>Impact of asphyxia on organ function</u>

Some organs (liver, kidney) can tolerate anoxia (no oxygen) for 15 to 60 min *(Piazza-2013)*. The human brain is an exception, being highly oxygen dependent. Interruption of oxygen supply to the brain for more than 5-10 seconds results in loss of consciousness *(Rossen-1943)*, and interruption for more than a few minutes causes irreversible damage *(Gooden-1994)*. The heart is more tolerant than the brain, although it can develop arrhythmias and heart block *(Fitz-Clarke-2018)*.

The average brain is about 2% of total body weight (1400 g, 3.08 lb), but receives 15% of resting cardiac output and consumes 20% of total body oxygen consumption (~49 ml/min of 250 ml/min) *(Fitz-Clarke-2018; Rink-Khanna-2011)*. The brain requires a very high level of metabolic activity in order to generate and (constantly) transmit enormous numbers of nerve signals *(Hoiland-AJP-2016)*. The brain's ability to process large amounts of oxygen over such a small tissue mass makes it especially vulnerable to decreases in arterial oxygen.

Humans have negligible capacity to downregulate metabolism because maintaining consciousness and cognitive function requires large amounts of energy. Unable to downregulate brain function when oxygen levels are low, humans instead experience a brief phase of cognitive decline followed

by abrupt loss of consciousness *(Fitz-Clarke-2018)*. Brain cells (neurons) no longer function when PO_2 at their surface is reduced below ~20 mmHg *(Nunn-1987-p473)*. The minimum PaO_2 that avoids cerebral hypoxia (inadequate brain oxygen) is 36 mmHg *(Nunn-1987-p474)*. Acclimatized mountaineers and divers remain conscious with PaO_2 as low as 20 mmHg *(Lindholm-UHM-2006)*, but an uncompensated person suddenly exposed to hypoxia is unlikely to remain conscious with PaO_2 less than ~27 mmHg (individual variation is considerable) *(Nunn-1987-p476)*.

In videos of strangulation by hanging, loss of consciousness is determined by inspecting the victim's face, voluntary movements, and body tone *(Sauvageau-AJFMP-2011;32:104)*. Between 11:08 [Dillard camera, 10:30] and 11:55 [Dillard camera, 11:17], Mr. Timpa made repeated efforts to lift his head and extend his neck. Mr. Timpa spoke his last audible words "Help me" at 11:40. The combination of lifting his head/extending his neck and audible speech signifies that his brain was still receiving sufficient oxygen to achieve voluntary muscle activity at 11:55. Between 12:01 and 13:33 [Dillard camera, 11:23 and 12:55], Mr. Timpa's head exhibited repeated rhythmical motion (detected by focusing on the left ear). The rhythmicity is consistent with repeated contraction of the sternomastoid, trapezius and serratus anterior muscles as occurs during agonal breathing *(Campbell-1970)*. At 13:23 [Dillard camera, 12:45], Mr. Timpa's left (upper) arm moves. Movement of the left arm occurred 1 min 28 sec (13:23 – 11:55 = 01:28) after Mr. Timpa's last voluntary contraction of his head and neck muscles. This left-arm movement, at 13:23 [Dillard camera, 12:45], signifies myoclonus-seizure type movement secondary to brain hypoxia *(Simon-Neurol-1999; Morris-JNNP-1998; Pesola-Westfal-1999; Sauvageau-JFS-2010)* and movements described during strangulation by hanging *(Sauvageau-Racette-Agonal-Seq-JFS-2007; Sauvageau-WG-eight-JFS-2010)*. The presence of the handcuffs makes it impossible to differentiate between decorticate posturing (characterized by extension of lower limbs and flexion of the upper limbs) and decerebrate posturing (characterized by extension of lower limbs and extension of the upper limbs) *(Sauvageau-WG-eight-JFS-2010)*. At 11:55 [Dillard camera, 11:17], Mr. Timpa exhibited his last voluntary muscle activity (lifting his head/extending his neck), after which he experienced apnea. With onset of apnea he no longer replenished his oxygen stores. As presented on **page 13** of my report, it took 1 min 32 sec for Mr. Timpa to completely use up his exchangeable oxygen stores after cessation of breathing. That is, at 13:27 (11:55 + 01:32 = 13:27), all of Mr. Timpa's exchangeable oxygen stores were totally depleted.

At 14:03 [Dillard camera, 13:25], Officer Dillard attempted to arouse Mr. Timpa by shaking his left shoulder with his left hand, immediately preceded by an officer calling out "Tony." At 14:18 [Dillard camera, 13:40], Officer Dillard made a more vigorous attempt to arouse Mr. Timpa. At 15:36, Officer Dillard lifted his left knee off Mr. Timpa's torso—that is, 3 min 41 sec after Mr. Timpa's last voluntary muscle activity and 2 min 9 sec after all of Mr. Timpa's exchangeable oxygen stores had been totally depleted (15:36 - 13:27 = 2.09). Mr. Timpa's face did not become visible until shortly before 16:00, when he was being moved to the gurney; at that time, he exhibited no facial features of animation (consciousness).

Autopsy

In her autopsy report, Medical Examiner, Emily Ogden MD, documented superficial contusions and abrasions on Mr. Timpa's head, trunk, and extremities. Dr. Ogden stated that examination of

the bulbar and palpebral surfaces of the conjunctiva and the laryngeal mucosa revealed no petechiae.

Some forensic pathologists place particular stress on detecting petechiae at autopsy in order to diagnose asphyxial death. Petechial hemorrhages result from the rupture of small vessels. Because capillary bleeding is not visible to the naked eye, petechiae are produced by small venules *(Clément-Sauvageau-2011)*. Some forensic pathologists claim petechiae are caused by increased pressure in neck veins together with injury of the inner lining of blood vessels (endothelial cells) secondary to low blood oxygen (hypoxia) *(Ely-Hirsch-2000)*. Drs. Ely and Hirsch (Chief Medical Examiner Office, New York) judge this misunderstanding of pathophysiology as the source for false claims that petechiae constitute "hallmarks of asphyxial deaths" *(Ely-Hirsch-2000)*. In reality, petechiae are caused by mechanical obstruction of venous return to the heart producing an increase in intravascular pressure that provokes overdistention and rupture of peripheral venules *(Clément-Sauvageau-2011)*.

Petechiae are more common in eyelids, sclera, and areas low in connective tissue. The frequency with which they are found following strangulation varies from series to series. In a recent large series of 383 asphyxia homicides from Finland, 187 dying by manual strangulation, petechiae were present in 59.89% (112/187) *(Wahlsten-2020)*. Rates of petechiae following strangulation in other series include the following: 21.6% (29/134) *(Plattner-2005)*, 21.5% (17/79) *(Zilkens-2016)*, 54% (22/41) *(Wilbur-2001)*, and 14.7% (15/102) *(Shields-2010)*. In his personal series of neck-pressure induced death, Dr. Bernard Knight, one of the world's foremost forensic pathologists, observed "the 'classical signs' … in slightly less than half the cases" *(Knight-Fatal-pressure-neck-2015-p361-389)*. Drs. Ely and Hirsch *(Ely-Hirsch-2000)* conclude "In and out themselves, they should not be regarded as supportive evidence of asphyxia; in a vacuum, conjunctiva and facial petechiae point to no particular cause of death."

Discussing autopsy findings in cases of asphyxia, Dr. Pollanen, chief forensic pathologist, Ontario, made the following observations: (a) "Rapid anoxial deaths are among the most enigmatic of all forensic entities that routinely confront the pathologist;" (b) "it is widely accepted that there are no specific or fully determinate signs of asphyxia at autopsy and that this diagnosis cannot be objectively made on the basis of postmortem appearances;" (c) "it is universally recognized that such lesions may be absent in some cases of subtle fatal homicidal neck compression" *(Pollanen-MSL-2001)*.

In her autopsy report, Dr. Ogden documented that Mr. Timpa's coronary arterial system and abdominal aorta was free of significant atherosclerosis, and infarction was not documented in his myocardium. Dr. Ogden claimed that Mr. Timpa's heart weight, 480 g, signified cardiac hypertrophy. On page 112 of her deposition, Dr. Ogden stated

> "So a normal heart, we would like to see it around 350 g. If someone of his size, I would even maybe give him up to about 400. But anything over 400, for sure, is too big."

On Page 123 of his deposition, Chief Medical Examiner, Dr. Jeffrey Barnard stated

> "He has a big heart"

and, on Page 138, Dr. Barnard, stated

> "480 is unequivocally big."

When considering oxygen dynamics in the body and tissue hypoxia, the question of whether Mr. Timpa's heart weight was elevated (or not) is of considerable importance. A heavier (or enlarged) heart requires a greater supply of oxygenated blood to satisfy its metabolic needs. When a person with a heavier heart engages in vigorous physical activity, he or she is vulnerable to under-perfusion of heart muscle (termed demand ischemia). At Dr. Ogden's deposition, Defense Attorney Ms. Gowin inquired "What is sudden cardiac death?" Dr. Ogden replied:

> "Usually an electrical disturbance, like an arrhythmia …I think he had some sort of arrhythmia" (page 110).

Dr. Ogden's answer is vacuous because every human who ever lived had an arrhythmia at the moment of death. Ms. Gowin also inquired "What are some of the risk factors that make it more likely that a person will experience sudden cardiac death?" (page 111). Dr. Ogden replied:

> "big heart; bad arteries."

On page 123 of his deposition, Dr. Barnard stated:

> "People with big hearts... are at risk for sudden death… frequently those areas of the heart are underperfused…you are predisposed to having ischemia at any point, frankly. Certainly increased when you're at increased exertion, and therefore you are at risk for a sudden cardiac event."

It is evident that the conclusions of Dr. Ogden and Dr. Barnard that Mr. Timpa's death was caused by a cardiac mechanism is predicated on their belief that his heart was hypertrophied.

Neither Dr. Ogden nor Dr. Barnard were asked about the reference standards they used for their judgments on heart size. Until recently, the most widely used standard was that generated by pathologists at the Mayo Clinic, resulting from 765 autopsies performed between 1960 and 1982 by Dr. Kitzman and associates *(Kitzman-1988)*. Subsequent to the depositions of Dr. Ogden and Dr. Barnard, a pathbreaking study entitled "Prevalence of abnormal heart weight after sudden death in people younger than 40 years of age" was published by Schoppen and colleagues on September 15, 2020 in the *Journal of the American Heart Association* *(Schoppen-2020)*.

The report of Schoppen and colleagues *(Schoppen-2020)* has two parts. First, they surveyed medical examiner offices and found that 52 centers reported 22 different methods to decide what was a normal heart weight. There was no consensus reference standard. Second, they reviewed all autopsies performed at the Cook County (Illinois) Medical Examiner's Office between 2014 and 2017 for individuals whose deaths were classified as *homicide, suicide*, or *accident*. This resulted in 3,398 cases who were certified as having a non-cardiac cause of death by the medical examiner. Previous studies consisted of far fewer cases, including the Mayo study by Kitzman and colleagues, which amassed only 765 hearts *(Kitzman-1988)*. The autopsies in the Schoppen study *(Schoppen-2020)* were from the ethnically diverse population of Chicago and Cook County, making the results more generalizable.

Schoppen and colleagues developed an equation based on age, sex, weight and height for estimating the upper 95% confidence interval of normal heart weight:

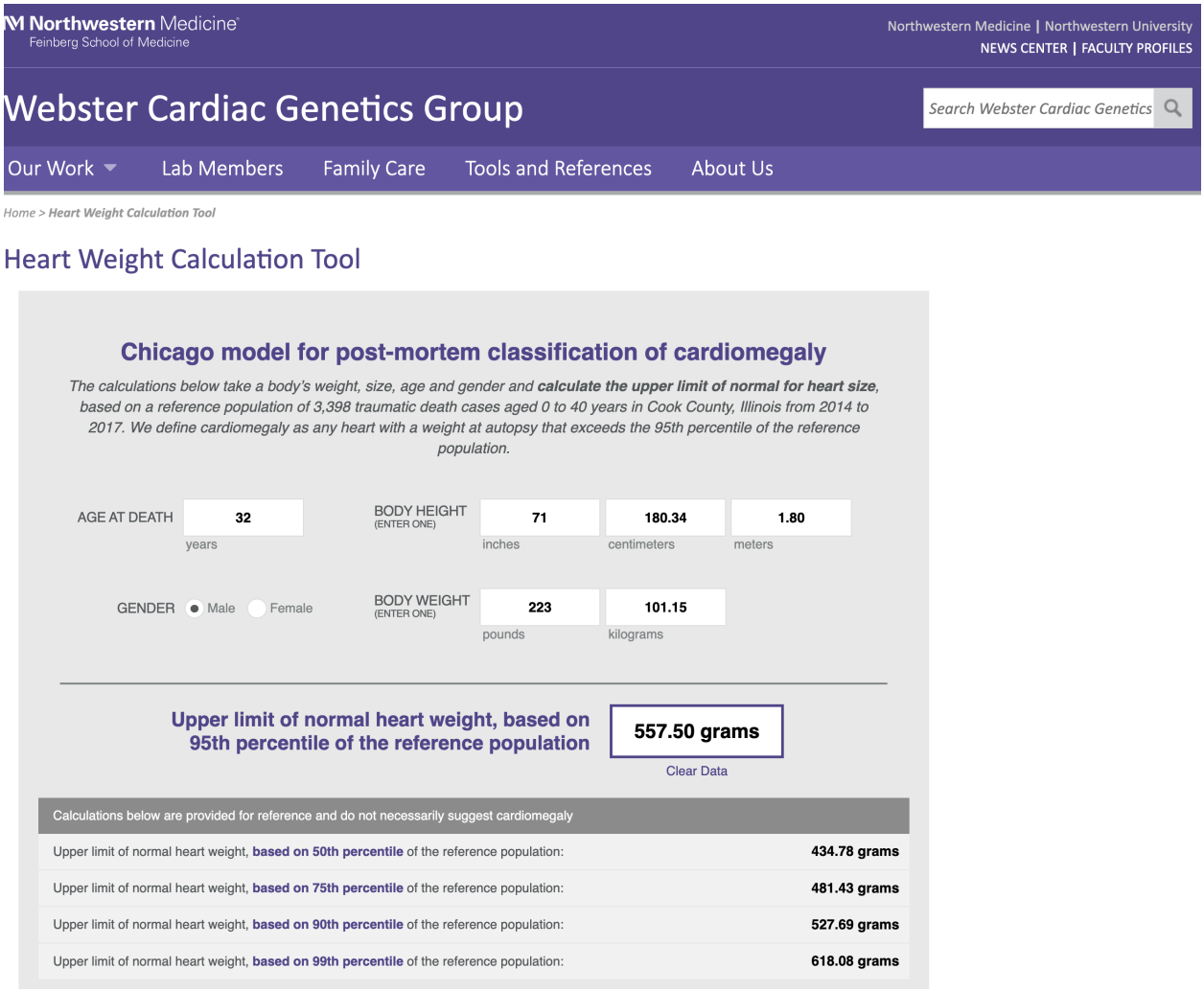
$$e^{2.88 - 0.12*\text{female} + 0.0065*\text{age} + 1.09h + 0.047w - 0.018w*h + 0.000092h*w2 - 0.0002w2 + 0.25}$$

where *age* refers to age at death; *h* is body height, and *w* is body weight. Schoppen and colleagues consider their equation more precise than tabular displays, as employed by Kitzman and

colleagues. Schoppen and colleagues provide a link to an online calculator to determine the threshold for 95th percentile threshold for cardiomegaly:

https://labs.feinberg.northwestern.edu/webster/heart_weight/

When Mr. Timpa's data—age 32 years, male sex, height 71 inches, weight 223 lb—are entered into the online calculator, the upper limit of normal (95th percentile) for heart weight is 557.50 g. Mr. Timpa's heart weighed 480 g, which is 77.5 g below the upper limit of normal.



When the depositions of Dr. Ogden and Dr. Bernard were taken (September 16, 2019 and February 13, 2020, respectively) the deponents were not in a position to know of the pathbreaking study that would be published in September 2020. They concluded that Mr. Timpa's heart weight represented cardiomegaly based on the best available scientific data at the time of the autopsy. The process of science is iterative, and when higher quality data based on larger sample sizes and more rigorous methodology are published, the new information supersedes pre-existing inferior information. To paraphrase John Maynard Keynes, when the facts change a scientist has to change his or her mind. Employing the most rigorous scientific information presently available, namely that of Schoppen and colleagues *(Schoppen-2020)*, it is clear that Mr. Timpa's heart weight was normal. As such,

conclusions reached by Dr. Ogden, Dr. Barnard, and other experts premised on a belief that Mr. Timpa's heart was enlarged can no longer be supported by the best science in cardiology.

Timing and cause of death

Following Mr. Timpa's arrival at Parkland Memorial Hospital at 11:12 PM, Dr. Alex Cohen pronounced him dead at 11:30 PM. The moment of biological death, however, occurred during the restraint. In her deposition, Dr. Ogden noted on page 123 that by the time Versed was administered (14:40), "his blood was not circulating. So, I think he was dead when they administered it." The time of death is often stated as if it occurs at one second in time. It is more accurate to describe death as a process, happening over time.

Mr. Timpa spoke his last audible words "Help me" at 11:40 and made his last voluntary movement of his head and neck at 11:55. Accordingly, Mr. Timpa was not experiencing cerebral hypoxia at 11:55 – signifying that his PaO_2 was 37 mmHg or higher *(Nunn-1987-p474)*. After 11:55, Mr. Timpa exhibited no further voluntary muscle contraction; the ability to engage in voluntary action and consciousness are lost when oxygen supply to the brain is stopped for more than 5-10 seconds *(Rossen-1943)*. Between 12:01 and 13:33 [Dillard camera, 11:23 and 12:55], Mr. Timpa's head exhibited repeated rhythmical motion of his head consistent with agonal breathing *(Campbell-1970)*. At 13:23 [Dillard camera, 12:45], Mr. Timpa's left (upper) arm moved, signifying myoclonus-seizure type movement secondary to brain hypoxia *(Simon-Neurol-1999; Morris-JNNP-1998; Pesola-Westfal-1999; Sauvageau-JFS-2010)*. Shortly before 16:00, Mr. Timpa was moved to the gurney, his face became visible and exhibited no facial features of animation (consciousness).

In the language of forensic medicine, the cause of death was asphyxia. Asphyxia is defined as failure of the cells of the body to receive oxygen combined with a high level of carbon dioxide consequent to inadequate respiratory exchange of gas with the environment *(Gooden-1993)*. In the absence of ventilation, the body attempts to sustain aerobic metabolism using oxygen stores in the lung and blood *(Fitz-Clarke-2018)*. When oxygen-based aerobic metabolism is no longer possible, anaerobic metabolism satisfies ATP requirements, but for no more than a few minutes, at which point critical pumps within the cell fail and cell death ensues *(Lipton-1999)*.

The mechanism of the asphyxia was compressive asphyxia, defined as a compromise of ventilation consequent to the application of an external force that interferes with movement of the chest and/or abdomen (in this case, downward pressure exerted by knees and hands pressing down on the torso) *(Nolan-EMJ-2020; Rutty-Madea-2021)*. When lethal hypoxemia or asphyxia is caused by trauma, the death is classified as a non-natural death *(Keil-Delbridge-Madea-2021)*.

Scientific questions raised during depositions

A. **Mr. Timpa's failure to say "I can't breathe":**

In depositions and reports, the police officers and experts placed particular emphasis on the fact that Mr. Timpa never stated "I can't breathe" or voiced a sense of difficulty in breathing. All of the depositions were taken before the Covid pandemic, and perhaps these individuals would be

less dismissive of Mr. Timpa's failure to vocalize a sense of dyspnea – the medical terminology for the symptom of difficulty in breathing – if the depositions were taken today.

In the early days of the Covid pandemic, many commentators were baffled by the failure of patients with life-threatening levels of low arterial oxygen (hypoxemia) to notice any difficulty with breathing. The *Wall Street Journal* considered it a medical mystery as to why "large numbers of Covid-19 patients arrive at hospitals with blood-oxygen levels so low they should be unconscious or on the verge of organ failure. Instead they are awake, talking—not struggling to breathe" *(WSJ-2020)*. The major scientific journal, *Science*, judged the lack of patient discomfort at extraordinarily low blood-oxygen concentrations as defying basic biology *(Science-2020)*. Dr. Levitan, with 30 years' experience of emergency medicine, wrote in *The New York Times* "A vast majority of Covid pneumonia patients I met had remarkably low oxygen saturations at triage—seemingly incompatible with life—but they were using their cellphones…they had relatively minimal apparent distress, despite dangerously low oxygen levels" *(NYT-2020)*.

These early reports were anecdotal, and the validity of the statements could not be verified. I have had a research interest in dyspnea since the 1980s and have published several investigations on different aspects of dyspnea *(Tobin-Chest-1983; Burki-ARRD-1988; Tobin-ArchIM-1990; Leung-AJRCCM-1997; Tobin-Gardner-PPICM-1998; Brack-AJRCCM-2002; Tobin-Handbook-2012; Laghi-Tobin-PPMV-2012)*. Accordingly, I was curious if the new phenomenon, dubbed "Happy Hypoxia" by the press, was real or fictitious. My colleagues and I undertook the first research study into silent hypoxemia (happy hypoxia) and published our findings in early months of the pandemic *(Tobin-AJRCCM-2020)*. We identified 16 patients who had arterial oxygen levels tensions (PaO_2) below 60 mmHg and who had communicated to a physician that they were not experiencing difficulty with breathing. In a healthy person, hypoxemia does not cause dyspnea until PaO_2 decreases to 60 mmHg (from a normal value higher than 90 mmHg). The primary sensors for hypoxemia are the carotid bodies, located within the carotid arteries. Responsiveness of the carotid bodies is influenced by several factors, especially the prevailing level of carbon dioxide in arterial blood ($PaCO_2$ – normal value, 35-45 mmHg). If $PaCO_2$ is 39 mmHg or lower, people will not experience dyspnea despite life-threateningly low levels of PaO_2 *(Moosavi-JAP-2003)*; 9 of our Covid patients had $PaCO_2$ values below 39 mmHg. The other 7 patients had $PaCO_2$ values greater than 39 mmHg (range, 41-49) and, despite extremely low PaO_2 values, did not complain of dyspnea – signifying that they truly had silent hypoxemia (happy hypoxia).

The phenomenon of silent hypoxemia (happy hypoxia), with failure of patients to complain of dyspnea despite near-fatally low levels of blood oxygen (and $PaCO_2$ values above 40 mmHg), demonstrates that defense experts have an inadequate understanding of the role of the brain's regulation of ventilation and its relationship to dyspnea. The failure of Covid patients to complain of dyspnea is understandable if one has a detailed grasp of pulmonary physiology *(Tobin-AJRCCM-2020)*. On page 79 of his deposition, defense medical expert and emergency room physician, Dr. Steven Bird, stated:

> "in my experience, people who don't have enough oxygen are not really struggling, they are not talking, they are becoming unresponsive."

When asked about clinical features in patients who are "not getting enough oxygen," Dr. Bird, stated (on page 120)

> "that's not someone who is moving, who is vocalizing. In my experience, it's not people who are able to move and vocalizing."

Both of Dr. Bird's statements have been proven scientifically invalid by the documentation of silent (happy) hypoxia during the COVID-19 pandemic *(Tobin-AJRCCM-2020)*. These invalid statements indicate that Dr. Bird's conclusions regarding the likelihood that Mr. Timpa did not experience hypoxemia (during restraint) are based on a flawed understanding of pulmonary physiology.

The danger of relying on dyspnea to warn of impending fatal hypoxemia has been known for decades and is especially treacherous for aircraft pilots: sudden loss of consciousness (without dyspnea) is frequently the first sign of life-threatening hypoxemia at high altitude *(Shaw-Front-Physiol-2021)*. Death from hypoxia without warning or complaint of dyspnea also occurs when oxygen in the atmosphere is displaced by an increased fraction of nitrogen. Nitrogen itself is an inert, non-toxic gas and without adverse effects on the body; it causes asphyxia and death by displacing environmental oxygen and decreasing the content of oxygen in alveolar sacs within the lung *(Gill-AJFMP-2002)*. Asphyxia deaths consequent to increased environmental nitrogen have been reported in several occupational settings, such as silos, ship holds, walk-in freezers, MRI repair areas, and especially in mines – famously in the Hartley Colliery disaster, when 204 men and boys asphyxiated from what miners call "black damp" or "stythe," which displaces environmental oxygen *(Gill-AJFMP-2002; Dunford-JEM-2009; Hendrick-BMJ-1992)*.

For years, police officers have employed the mantra "If he can speak, he can breathe." This phrase was repeated over and over by the officers involved in the death of Mr. George Floyd. The timeline in the Floyd case highlights the danger and false sense of security engendered by this mantra. Twenty-two seconds after Mr. Floyd's last spoken words, he experienced vigorous extension of his right lower extremity, representing myoclonus-seizure type movements and signifying severe hypoxic brain injury. The 22-sec interval highlights the rapidity with which a person can transition from being able to speak words to suffering (fatal) hypoxic brain injury. Police officers present during the restraint of Mr. Timpa also expressed the highly dangerous concept "If he can speak, he can breathe."

Medical Examiner, Dr. Emily Ogden, performed the autopsy on Mr. Timpa. At several junctures during her deposition, she was asked why she had concluded that Mr. Timpa did not experience significant asphyxia. On page 24 of her deposition, Dr. Ogden stated:

> "based on the fact that he is yelling, he is fighting, he is moving, I don't think that he experienced significant asphyxia."

On page 72, Dr. Ogden is asked why she thought Mr. Timpa's death was not caused by asphyxia, and replies

> "Because of the fact that I can see him yelling and moving for the majority of the restraint."

On page 11, Dr. Ogden was asked what she saw that indicated that Mr. Timpa could breathe and answered:

> "the fact that he is yelling."

Dr. Bird also claimed that Mr. Timpa's ability to speak excluded the likelihood of mechanical or positional asphyxia. On page 58 of his deposition, Dr. Bird remarked:

> "Mr. Timpa was vocalizing… He was able to talk."

On page 61, Dr. Bird was asked to describe the features in patients who are not able to respirate, and answered:

> "They are not able to vocalize. They are not struggling like Mr. Timpa was. They are typically unresponsive or nearly unresponsive."

On page 145, Dr. Bird was asked "Can you rule out mechanical or positional asphyxia as a cause of death in this case?" Dr. Bird replied

> "Yes," adding "We know that it doesn't happen. That's been a theory disproved that occurs during restraint. Furthermore, he's vocalizing, talking, ranting, moaning throughout most of it."

This claim by Dr. Bird that asphyxia "doesn't happen" would be a surprise to the families of the 96 spectators who died from asphyxia in the Hillsborough disaster *(Nolan-EMJ-2020)*. The treacherous consequences of a belief that ability to speak signifies no difficulty with breathing is not based solely on the universally witnessed example of Mr. George Floyd vocalizing repeatedly before experiencing clinical signs of fatal hypoxic brain injury 22 seconds after his last spoken words. In reality, the false sense of security engendered by this erroneous belief has been recognized for decades. Patients who suffer from disorders that produce upper airway obstruction are able to speak until the airway aperture narrows to 15% of its original dimensions—an area little bigger than a pinhead *(Wain-2003; Biswas-Chest-2018; Biswas-Chest-2018)*. A person can be speaking one moment, and, a few seconds later, lose consciousness because the supply of oxygen to the brain suddenly plummets. Following Mr. Floyd's death, I have not seen a medical expert claim that a detainee's ability to speak can be taken as evidence that he or she is not at risk of death from alveolar hypoventilation.

There are additional scientific factors, specific to Mr. Timpa, that influence his failure to express difficulty with breathing. Dyspnea is ultimately sensed at the level of the cerebral cortex and involves a patient communicating the sensation of a lack of air or difficulty with breathing *(Tobin-ArchIM-1990)*. Dyspnea arises from signals generated by chemoreceptors, mechanoreceptors or other receptors, which are transmitted to the respiratory centers in the medulla oblongata (brainstem) and, in turn, this heightened respiratory drive is transmitted from the brainstem to the top of the brain (the cerebral cortex) *(Tobin-ArchIM-1990; Tobin-Gardner-PPICM-1998; Tobin-AJRCCM-2020)*. It is this "corollary discharge" from the brainstem to the cerebral cortex that produces the noxious sensation of dyspnea *(Banzett-Compr-Physiol-2021)*.

Localization of the particular areas of the cerebral cortex involved in the perception of dyspnea is an area of intense research, employing blood oxygen level dependent (BOLD) functional magnetic resonance imaging (fMRI) *(Evans-Banzett-J-Neurophysiol-2002)*, positron emission tomography (PET scan) *(Brannan-PNAS-2001; Liotti-PNAS-2001; Peiffer-AJRCCM-2001)*, and other techniques. Scientific investigations have repeatedly shown that the insula, anterior cingulate cortex, amygdala and limbic system are pivotal areas in sensing the noxious stimulus of dyspnea *(Banzett-Compr-Physiol-2021)*. These anatomical areas are also involved in the sensing and processing of pain *(Wager-NEJM-2013; Brown-PLoS-2011; Tracey-Neuron-2007; Hofbauer-Anesthesiol-2004; Apkarian-EJP-2005)*. For more than 60 years, researchers have highlighted the similarities between dyspnea and pain *(Comroe-MCCD-1956)*, and neuroimaging investigations over the last 30 years have provided robust concrete evidence of the overlap.

Mr. Timpa had a long-standing history of mental illness and he told the 911 dispatcher that he suffered from schizophrenia. For over a century it has been recognized that psychiatric patients have decreased awareness of stimuli that induce pain *(Singh-JPP-2006)*. Numerous case reports provide vivid accounts of patients with psychiatric conditions, including schizophrenia spectrum disorder, exposed to experiences that normally cause considerable pain – placing a hand in a flaming fire *(Virit-2008)*, bowel perforation and abscess formation *(Murakami-Lancet-2010)* – and yet the patients did not communicate any sense of pain or demonstrate expected clinical signs on physical examination. MRI studies in patients with schizophrenia spectrum disorder reveal the largest volumetric decreases in paralimbic areas of the cerebral cortex, particularly in the anterior cingulate and paracingulate gyri and the insula *(Goldstein-AGP-1999)*—the anatomical areas involved in perception of pain and in perception of dyspnea *(Tracey-Neuron-2007; Banzett-Compr-Physiol-2021)*.

In a 1955 landmark study in the *New England Journal of Medicine*, Dr. Marchand reported on 51 patients with mental disorders who experienced acute myocardial infarctions *(Marchand-NEJM-1955)*. After exclusion of cases of sudden death and consideration of presenting symptoms, painless myocardial infarction was documented in 82.5% of the patients. In another 1950s *NEJM* report of patients with psychiatric disease, pain was absent as a presenting symptom in 41.3% of patients with fracture of the femur *(Marchand-NEJM-1959)*.

In a survey of the world literature on decreased pain sensitivity in patients with schizophrenia, Stubbs and colleagues *(Stubbs-Pain-2015)* performed a meta-analysis of experimental studies investigating pain among patients with schizophrenia spectrum disorder versus healthy controls. The primary outcome was a pooled composite of pain threshold and pain tolerance. Across 17 studies, patients with schizophrenia spectrum disorder (n=387; age, 30.7±6.9 years; 68.1% male) had a highly statistically significant increase in their pain threshold or pain tolerance versus healthy controls (effect size=0.583; 95% CI, 0.212-0.954; p=0.002). When investigators excluded patients being treated with antipsychotic medications, the findings were similarly significant.

The phenothiazine, chlorpromazine (Thorazine), was the first antipsychotic drug, also termed neuroleptic. Thorazine's discovery was the result of serendipitous observations by a French surgeon, Henri Laborit, who was searching for a better anesthetic *(Lehmann-CJP-1997)*. As other neuroleptics were introduced, anesthesiologists combined a neuroleptic with an analgesic to achieve what they termed neuroleptanalgesia, employed during minor surgical procedures and endoscopy *(De-Castro-1959)*. Neuroleptanalgesia gained considerable popularity because it achieved a state of quiescence, psychic indifference and freedom from pain, while the patient remained conscious and fully rousable *(Stevenson-Thorax-1972)*. When I started to train in bronchoscopy in 1978-79, neuroleptanalgesia was widely employed *(Keller-Chest-1975)*. (Other approaches to conscious sedation have since replaced neuroleptanalgesia.) It is, thus, hardly surprising that researchers have demonstrated that psychiatric medications achieve a considerable decrease in pain.

To determine the effects of antipsychotic agents in the treatment of acute and chronic pain, Dr. Seidel and colleagues undertook a Cochrane meta-analysis of five randomized double-blind controlled trials *(Seidel-Cochrane-2013)*. Quantitative analysis of these studies showed a significant reduction of mean pain intensity after administration of the antipsychotic compared to

placebo or another active compound: weighted mean difference -1.78 (95a% CI -2.71 to -0.85) and relative risk 0.43 (95% CI 0.25 to 0.73).

In addition to the above general effects of psychiatric medication on sensitivity and tolerance of pain, Dr. Ogden's autopsy report includes a list of 5 specific psychiatric medications prescribed for Mr. Timpa. These agents have different pharmacological actions on circuits within the neural architecture that regulate perception of sensory stimuli and cognition – key factors in expressing a complaint of dyspnea. I will discuss the pharmacological actions of each agent.

One, bupropion (Wellbutrin) is a noradrenaline and dopamine reuptake inhibitor, which also acts as an inhibitor of nicotine a4b2 receptor and 5-hydroxytryptamine (serotonin) receptors *(Costa-DMR-2019)*. Bupropion is used in the treatment of major depressive disorder and other mental disorders. In a double-blind randomized trial in patients with chronic obstructive pulmonary disease (COPD), bupropion increased the rate of smoking cessation and decreased symptoms of craving and withdrawal *(Tashkin-Lancet-2001)*. In another randomized double-blind trial that employed fMRI in nicotine-dependent cigarette smokers, smokers treated with bupropion exhibited significantly greater reductions in activation of the anterior cingulate cortex and other brain regions responsible for emotional and cognitive appraisal while resisting craving *(Culbertson-AGP-2011)*—anatomical areas involved in the sensing of dyspnea.

Two, lisdexamfetamine (Vyvanse), an inactive prodrug converted in the body to pharmacologically active dextroamphetamine, is used in the treatment of attention-deficit/hyperactivity disorder (ADHD) and other mental disorders *(Coghill-CNS-Drugs-2014)*. In a randomized crossover trial employing fMRI in adults with ADHD, lisdexamfetamine produced potentiated affective encoding in the amygdala and reduced psychophysiological interactions with the left inferior frontal gyrus *(Schulz BPCNN- 2018)*—anatomical areas involved in the sensing of dyspnea.

Three, guanfacine (Intuniv) is a selective norepinephrine α_{2A}-adrenoceptor agonist, used in the treatment of ADHD *(Bernknopf-AFP-2011.)*, and widely used off-label for several mental disorders that involve impaired functioning of the prefrontal cortex, such as substance abuse, schizotypic cognitive deficits, and traumatic brain injury *(Arnsten-NLM-2020)*. In healthy adults undergoing fMRIs, guanfacine produced activation of the thalamo-frontal-striatal network, including the bilateral dorsolateral prefrontal cortex, a region that adjusts control over thalamic nuclei, cingulate motor areas *(Clerkin-BP-2009)*, and other areas involved in the sensing of dyspnea.

Four, naltrexone (Vivitrol) is an opioid receptor antagonist with greatest affinity for mu-opioid receptors, but also occupies most kappa-opioid receptors (which interact with the stress-related dynorphin system) and has some delta-opioid-receptor action *(Kjome-SA-2011; Elton-ACER-2019)*. Naltrexone is used in the treatment of alcohol dependence, opioid dependence, and impulse-control disorders *(Kjome-SA-2011; Elton-ACER-2019)*. Several studies have demonstrated that naltrexone is successful in the treatment of chronic pain, acting through modulation of glial cells and release of inflammatory mediators in the central nervous system *(Kim-CPHR-2020)*. In a study employing fMRI in alcohol-using men, naltrexone altered intrinsic functional connectivity

in portions of the rostral anterior cingulate and medial prefrontal cortex *(Elton-ACER-2019)*—areas involved in the sensing of dyspnea.

Five, desvenlafaxine (Pristiq) is a serotonin-norepinephrine reuptake inhibitor used in the treatment of major depressive disorder and other mental illnesses. Desvenlafaxine and related anti-depressants have been shown to demonstrate reduced functional connectivity in a neural network associated with pain *(Sheline-Lancet-Psychiatry-2019)*. In a double-blind placebo-controlled trial employing fMRIs, desvenlafaxine produced decreased functional connectivity within the thalamo-cortico-periaqueductal network that is involved in the experience of pain—a region referred to as the "neurologic signature of physical pain" *(Wang-Lancet-Psychiatry-2019)*. Specific anatomical areas affected by desvenlafaxine include the insula, the cingulate cortices, and other areas that play a critical role in cortical sensing of dyspnea *(Banzett-Compr-Physiol-2021)*.

The above 5 psychiatric medications prescribed to Mr. Timpa, with multiple actions on regions of the brain critically involved in the perception of pain and in perception of dyspnea, provide a pharmacological basis—in addition to the already discussed physiological basis—for why Mr. Timpa did not complain of dyspnea.

B. **Mr. Timpa's grunting and its significance**

Comments by factual witnesses and expert witnesses: During the course of their depositions, two police officers involved in the restraint of Mr. Timpa, Officer Dustin Dillard and Corporal Raymond Dominguez, agreed that Mr. Timpa was grunting (page 17 of Dillard deposition and page 63 of Dominguez deposition).

On page 5 of his report, defense use-of-force expert, former police officer Mr. Craig Miller, wrote:

> "The BWCs show that Mr. Timpa continued to strain against the officers' restraint…mutter, grunt, and yell things."

Later in his report, Mr. Miller repeated that Mr. Timpa was grunting. On page 5, Mr. Miller wrote

> "At the 01:04 mark in Dillard's BWC video… Mr. Timpa grunts a response"– (Footnote 11: Dillard BWC 01:04-01:10).

On page 6, Mr. Miller wrote

> "He grunted and moved his head from side to side; his grunting tapers off and is last heard at 12:19 (Footnote 19: Dillard BWC at 11:12-11:55).

On page 6 of his report, defense medical expert, Dr. Steven B. Bird, MD, wrote "Mr. Timpa can be heard yelling, grunting, and making other vocalizations throughout the video." On page p116-117 of his deposition, the following exchange occurs:

> Q. During that minute that we just played, did you, do you think, did you hear any labored breathing by Mr. Timpa during that entire portion?
>
> A. As I said in my report, I think there was kind of grunting and moaning kind of throughout.
>
> Q. I guess, would you consider that labored breathing?
>
> A. I don't think I can opine.

Medical significance of grunting: A grunt is a noise heard during expiration. It is preceded by a pause of varying duration at the end of inspiration during which time the vocal cords (glottis) are

closed. As the vocal cords open, the sudden release of air under positive pressure produces the noise (the grunt).

Physicians taking care of adult patients today rarely mention grunting in their clinical notes. This contrasts with descriptions of pneumonia in the pre-antibiotic era. Grunting was described as a key feature of lobar pneumonia in classic medicine texts. In the foundational textbook of internal medicine, Dr. William Osler wrote the following in 1892: "The type of breathing in pneumonia is peculiar and almost distinctive. The inspirations are short and superficial. Expiration is often associated with a short grunt" *(Osler-1892)*. In his 1935 textbook, Dr. Russell Cecil wrote the following about the general appearance of patients with lobar pneumonia: "[Breathing is usually labored on account of the pain in the side. There is a dry hacking cough, and the nostrils the late with inspiration.] Frequently the patient gives a little grunt with each expiration *(Cecil-1935)*.

The symptom of dyspnea is subjective and depends on self-evaluation by patients *(Tobin-ArchIM-1990)*. ICU patients have difficulty in communicating because of an endotracheal tube in the airway, and caregivers pay less attention to dyspnea than to pain *(Banzett-Tobin-PPMV-2012)*. In a prospective study of patients being weaned from mechanical ventilation, about half of physicians and nurses substantially underestimated the level of patient dyspnea compared with self-reports by patients *(Haugdahl-AJRCCM-2015)*. Under-detection and underestimation of dyspnea also occurs with end-of-life care *(Campbell-JPM-2009)*. To increase caregiver awareness of dyspnea and improve its treatment, Dr. Campbell developed a Respiratory Distress Observation Scale (RDOS) which scores behavioral clues of respiratory distress in patients unable to report dyspnea *(Campbell-JPM-2010)*. RDOS consists of a scale with 8 observer-rated variables, one of which is grunting at end-expiration. (The other 7 variables are heart rate, respiratory rate, accessory muscle use, paradoxical breathing, restlessness, nasal flaring, and facial fear.) Dr. Campbell's research underscores that Mr. Timpa's grunting (noted by two police officers and two defense experts) was an objective indicator of respiratory distress.

Grunting is a cardinal finding in newborn babies with hyaline membrane disease (respiratory distress syndrome), and some of the most sophisticated investigations into the pathophysiology of granting have been conducted by neonatologists. Harrison and colleagues *(Harrison-Pediatrics-1968)* obtained detailed measurements of airflow, intrathoracic pressure, and abdominal muscle electromyography (EMG) in 22 infants with severe hyaline membrane disease during the first 16 hours of life. All infants exhibited rapid breathing, cyanosis and expiratory grunting. Inspiration was invariably rapid, during which time transpulmonary pressure reached a negative value of -12 cm H_2O. This was followed by an end-inspiratory pause during which the glottis remained closed. Expiration was active, and EMG recordings revealed contraction of the abdominal muscles during early expiration. Transpulmonary pressure switched from a negative value of -12 cm H_2O during inspiration to a mean positive value of +3.4 cm H_2O. Grunting was not heard during this initial phase. With opening of the vocal cords, air was rapidly expelled and this period coincided with audible grunting. Grunting ceased once the air had been expelled. The cycle together with grunting repeated itself over and over.

Grinman and Whitelaw *(Grinman-Chest-1983)* employed a pneumotachograph, magnetometers, esophageal pressure, and gastric pressure measurements in a 63-year-old man with generalized muscle weakness and progressive shortness of breath, who made grunting noises with each breath.

They found the same physiological pattern that Harrison and colleagues *(Harrison-Pediatrics-1968)* described in infants with hyaline membrane disease. In addition, Grinman and Whitelaw *(Grinman-Chest-1983)* observed a double positive abdominal-pressure wave in their patient. This pattern of a double positive abdominal-pressure wave is also seen in healthy people when they increase minute ventilation to 50-60 L/min during exercise (compared with 6 L/min at rest). As such, expiratory grunting is a marker of the severity of stress under which the respiratory system is operating.

Clergue, Whitelaw and colleagues *(Clergue-Anesthesiology-1995)* employed the same methodology to study five patients who exhibited expiratory grunting following elective cardiac surgery. The pattern of respiratory muscle activity contraction was similar to that reported by Harrison and colleagues in infants *(Harrison-Pediatrics-1968)* and that described by Grinman and Whitelaw *(Grinman-Chest-1983)*.

It is possible that grunting promotes an increase in cardiac output at a time of patient distress. In patients experiencing cardiac arrest during coronary angiography, the sequence of changes in intrathoracic pressure during cough produced higher mean systolic aortic pressure, 139.7±3.8 mm Hg, than during external CPR, 60.7±5.1 mm Hg—sufficient to achieve successful resuscitation from ventricular fibrillation *(Criley-JAMA-1976)*. Although equivalent studies have not been performed during grunting, the pattern of change in intrathoracic pressure during a grunt is similar to that during cough.

From the above, it is clear that detection of grunting signifies respiratory distress in infants and adults. The two police officers that endorsed Mr. Timpa's grunting and the two defense experts who volunteered that Mr. Timpa was grunting most likely did not recognize that grunting is an important objective indicator of significant respiratory distress, which countered other opinions they expressed about Mr. Timpa.

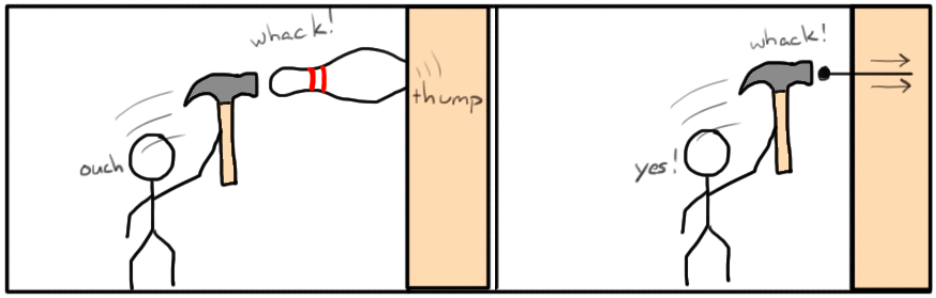
C. **Research conclusions of Dr. Chan and colleagues**

In their depositions, Dr. Ogden, Dr. Barnard, Dr. Bird and Dr. Kroll accept uncritically the findings of Dr. Chan and the San Diego research group at face value, and concur with Dr. Chan's claim that decreases in lung function consequent to kneeling on the back of a prone subject are not of a magnitude to be clinically significant. Both the design and interpretation of Dr. Chan's experiments contain several fundamental flaws. I will discuss three major design flaws – any of one of which is sufficient to render the entire Chan thesis otiose.

The first fatal flaw is embedded as a foundation stone in all the Chan experiments. This flaw involves a basic principle of physics, wherein pressure is defined as the amount of force exerted over a particular area:

$$P = \frac{F}{A}$$

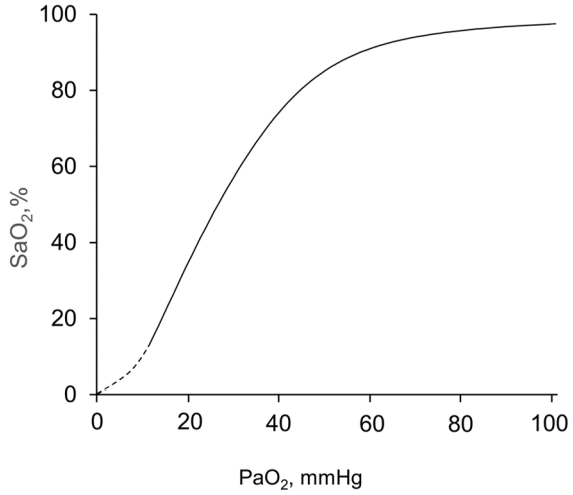
To generate a large amount of pressure, you can either exert a large amount of force or decrease the area over which the force is being exerted. The concept is illustrated in the cartoon below.



Hammering a bowling pin into a wall is not effective because the base of the pin has a broad area. Hammering a nail into a wall is effective because the base of the nail is concentrated on a very small area (its sharp tip). All of the force between the nail and the wall is concentrated on a very small area at the sharp tip of the nail. With the bowling pin, the area touching the wall is much larger, and, therefore, the force is much less concentrated.

Given that effective pressure is force divided by area, effective pressure increases in direct proportion to the decrease in area over which the force is applied. The article from Chan's group, "Savaser-Chan et al. The effect of the prone maximal restraint position with and without weight force on cardiac output and other hemodynamic measures. J Forensic Leg Med 2013;20:991," includes a figure of a research subject lying prone with two 45 lb weights placed on his back *(Savase-Chan-JFLM-2013)*. From the photograph, it is evident that the investigators used a 45-pound Olympic barbell plate, the dimensions of which are 17.5 inches by 17.5 inches, yielding a cross-sectional area of 240.5 square inches. The dimensions of my knee are 6 inches by 4 inches, yielding a cross-sectional area of 24.0 square inches. That is, the cross-sectional area of Olympic barbell plate is 10 times greater than the cross-sectional area of a knee. Because effective pressure is force divided by area, the pressure exerted by an Olympic barbell plate is 10 times less than the pressure exerted by a knee. In their articles, Dr. Chan and colleagues underestimate the pressure exerted by a knee by a factor of 10.

The second flaw relates to Dr. Chan's claim that observed decreases in lung function (consequent to some experimental condition) are not clinically significant because they do not produce significant decreases in oxygen saturation as measured by pulse oximetry. Because the oxygen-dissociation curve has a sigmoid shape, arterial oxygen saturation (SaO_2) is an inherently insensitive physiological tool for detecting changes in body oxygenation in healthy subjects *(Tobin-JAMA-1990)*. This problem is compounded by the use of a pulse oximeter to estimate oxygen saturation (SpO_2) as opposed to the direct measurement of SaO_2 by an arterial blood gas, because the 95% confidence limits of pulse oximetry ranges between +4% and -4% *(Jubran-Tobin-PPICM-1998)*.



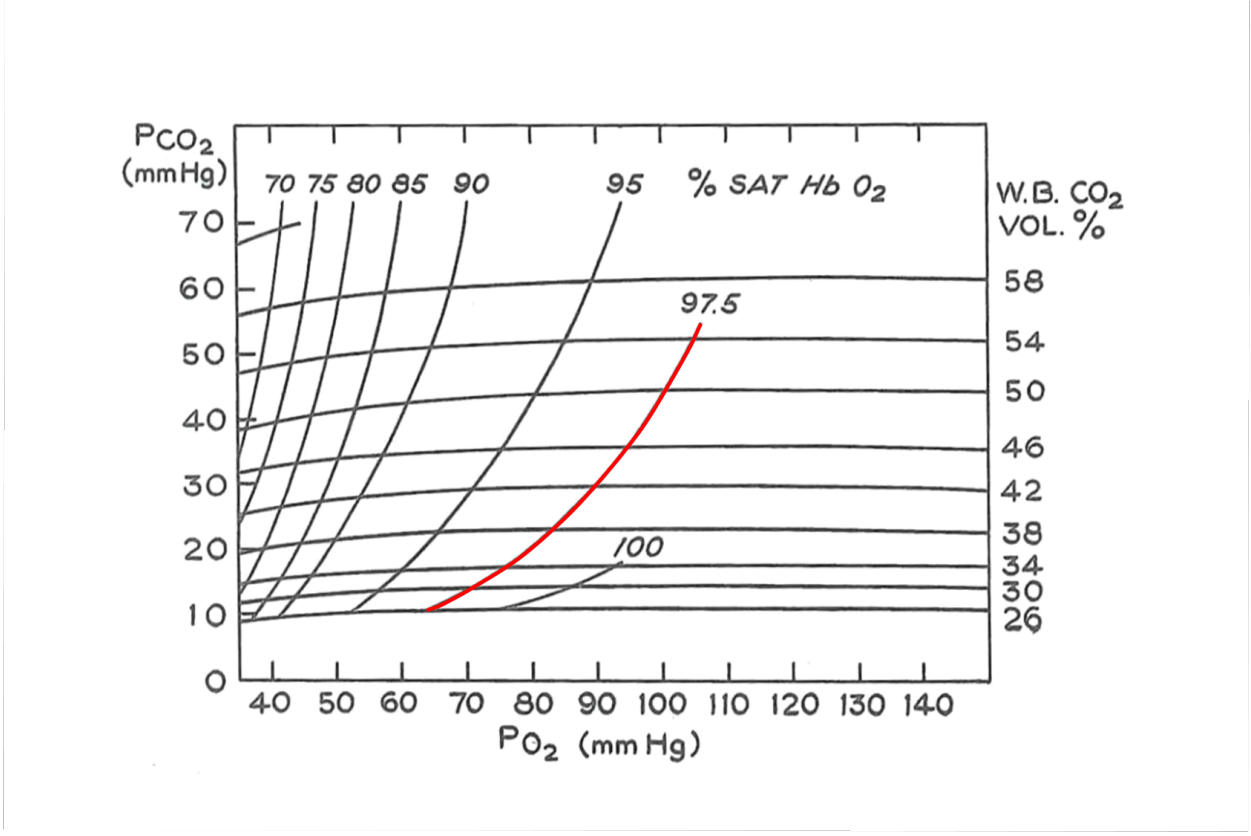
In a healthy person (as were the Chan subjects), the pressure of arterial oxygen (PaO_2) approximates 100 mmHg. This pressure constitutes the proximal force that drives oxygen molecules from the bloodstream into tissues and cells according to Fick's law:

$$\dot{V}gas = D_M \times (P1 - P2)$$

where the rate of gas transfer across a tissue plane or membrane (Vgas) is directly proportional to the difference in partial pressures of the gas across the two sides of the membrane (P1 – P2) and the membrane's diffusing capacity (D_M). A normal value of PaO_2 100 mmHg is accompanied by SaO_2 97.5% *(Severinghaus-JAP-1979)*. Because of the sigmoid shape of the oxygen-dissociation curve and because PaO_2 100 mmHg and SaO_2 97.5% are located on the upper flat portion of the curve (at considerable distance from the curve's upper inflection point, and well to the right from the inflection), a 30-mmHg fall in PaO_2 (from 100 mmHg to 70 mmHg) will be accompanied by a decrease in SaO_2 of only 3.5% *(Severinghaus-JAP-1979)*. Given that a 3.5% change in SaO_2 falls within the +4% to -4% range of the 95% confidence limits of pulse oximetry, a 30-mmHg fall in PaO_2 (from 100 mmHg to 70 mmHg) may produce little or no change in SpO_2 on a pulse oximeter *(Tobin-JAMA-1990; Jubran-Tobin-PPICM-1998)*. A 40-mmHg fall in PaO_2 (from 100 mmHg to 60 mmHg) will be accompanied by a 6.5% decrease in SaO_2 *(Severinghaus-JAP-1979)*, which again falls within the +4% to -4% range of the 95% confidence limits of pulse oximetry and may also produce little or no change in SpO_2 on a pulse oximeter. With a clear understanding of the oxygen-dissociation curve together with knowledge of the operating 95% confidence limits of the technology embedded within a pulse oximeter, Dr. Chan's claim that lack of a significant decrease in SpO_2 (on pulse oximetry in healthy subjects) constitutes proof that significant deterioration in ventilatory function (and the driving pressure that moves oxygen molecules out of the bloodstream and into peripheral tissues) did not occur is not only misleading but potentially treacherous.

The insensitivity of pulse oximetry in detecting deterioration in body oxygenation is compounded by right or left shifts in position of the oxygen-dissociation curve. The curve shifts to the right with acidosis, increase in PCO_2, increase body temperature, and certain hemoglobinopathies *(Jubran-Tobin-PPICM-1998)*. The curve shifts to the left with decreases in these physiological factors, and

also with fetal hemoglobin and carbon monoxide intoxication. A leftward shift in the curve, which is inevitable with a decrease in arterial carbon dioxide pressure ($PaCO_2$), means that large decreases in PaO_2 will not cause a decrease in SaO_2 and thus will not be detected by SpO_2 recordings on a pulse oximeter. In one of the experiments conducted by the Chan group *(Sloane-Chan-FSI-2014)*, the researchers measured carbon dioxide at the end of an exhaled breath, termed end-tidal PCO_2. When the subjects were seated at rest, $P_{ET}CO_2$ was 40.3±2.6 (SD) mmHg. Fifteen minutes after the subjects had performed 6-10 min of exercise on a cycle ergometer (reaching 85% of maximum heart rate), they were placed in the Prone Maximal Restraint Position (PMRP)—prone with wrists secured together behind the back and ankles drawn up near the wrists, without weights superimposed on the back. In this position, Chan and colleagues recorded a statistically significant decrease in $P_{ET}CO_2$ to 32.0±3.2 mmHg. Pulse oximeter measurements of SpO_2 in the sitting and maximum-restraint settings were respectively 96.5±1.3% and 95.7±1.7%; that is, a decrease of 0.8%, which is $^1/10^{th}$ of the observed 8.3 mmHg decrease in $P_{ET}CO_2$ *(Sloane-Chan-FSI-2014)*.



Understanding the consequences of a shift in the oxygen-dissociation curve is best grasped by employing the Rahn-Fenn diagram, one of the canonical graphs in pulmonary physiology *(Rahn-Fenn-1955)*. The Rahn-Fenn diagram plots PO_2 and PCO_2 against SaO_2. On the isopleth for SO_2 97.5% (close to SpO_2 96.5% at sitting baseline in the Sloane-Chan experiment), PCO_2 40.3 mmHg lines up with PO_2 99 mmHg and PCO_2 32.0 mmHg lines up with PO_2 92 mmHg. That is, PaO_2 can decrease by 7.0 mmHg solely as a result of a leftward shift in the oxygen-dissociation curve (consequent to 8.3 mmHg decrease in PCO_2), and this decrease in PaO_2 will not be accompanied by any decrease in SpO_2 on pulse oximetry. The lengthy discussion in the Sloane-Chan article

(Sloane-Chan-FSI-2014) makes no mention of the masking effect of the decrease in $P_{ET}CO_2$ and the false sense of security engendered by the mere 0.8% decrease in SpO_2 on pulse oximetry.

The third fatal flaw present in all of the Chan articles again relates to the claim that the observed decreases in lung function consequent to some experimental imposition – turning a subject prone or placing a weight on the back – are not clinically significant. This claim has been very persuasive, and Dr. Barnard, Dr. Bird and Dr. Kroll repeated this message uncritically in their depositions. The magnitude of the decrease in lung function varies with the experimental design in different studies. In the experiment by Michalewicz-Chan et al, placement of a weight of 102.3 kg (225.53 lb) on the back produced a 30.3% decrease in maximum voluntary ventilation (MVV, from 156±38 L/min at baseline sitting to 109±28 L/min while prone with weight), about which the authors wrote "the decrease in MVV was of no clinical importance in these subjects" *(Michalewicz-Chan-AEM-1997)*. In a study with a similar design, Cary and coworkers found that placement of sand-filled bags weighing 75 kg (165.35 lb) and placed evenly across the back of subjects restrained prone produced a 33.0±l.2 % decrease in MVV*(Cary-J-Physiol-2000)*. In none of their articles do Chan and colleagues contemplate the impact of a 30-33% decrease in lung function when a person is faced with an increase in ventilatory demands.

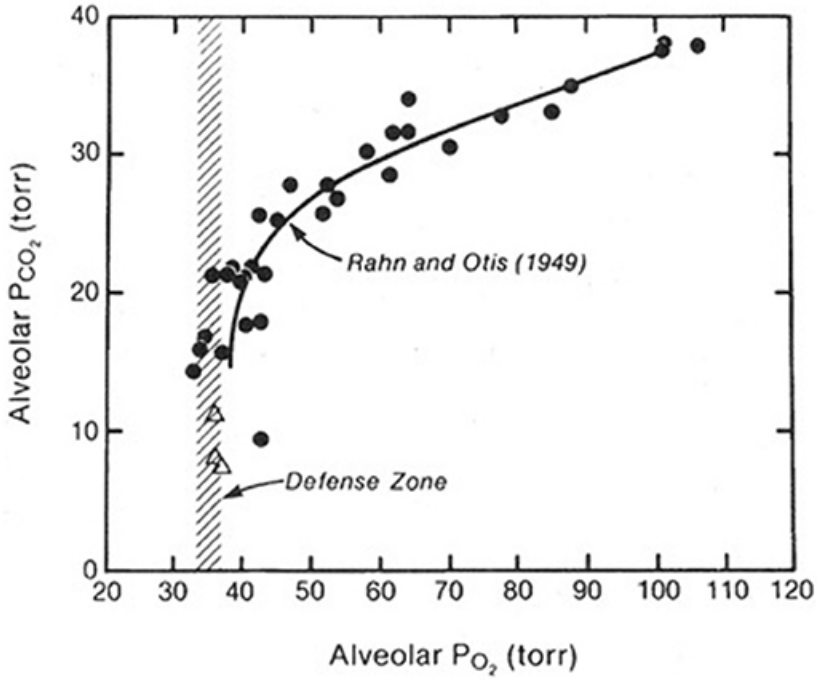
When a patient or healthy person is faced with constrained alveolar ventilation, survival depends on maintaining a viable level of arterial oxygenation. My colleagues and I have undertaken numerous physiological investigations on patients experiencing near-fatal acute ventilatory failure at the point of weaning from mechanical ventilation *(Yang-Tobin-NEJM-1991; Jubran-Tobin-AJRCCM-1997; Esteban-Tobin-NEJM-1995; Laghi-Tobin-AJRCCM-2003; Parthasarathy-Tobin-JAP-2007)*. These research studies have elucidated the precise mechanical impediments and the role of respiratory muscle performance in defending against alveolar hypoventilation. Ethical considerations prohibit investigators from studying the evolution of progressive lowering of oxygen levels to the point of death in human subjects. Over the last 40 years, however, a number of rigorous physiological studies have been conducted in mountaineers who have reached the top of Mt. Everest without supplemental oxygen.

Before 1978, it was widely believed that it would be impossible to reach the Everest summit (8848 m, 29,029 feet) without supplemental oxygen. That assumption was nullified by Italian Reinhold Messner and Austrian Peter Habeler who reached the summit on May 8, 1978 without supplemental oxygen *(Lankford-WEM-2021)*. Since then, about 200 persons (less than 4% of climbers) have reached the Everest summit without supplemental oxygen *(Grocott-NEJM-2009)*. As ascent progresses, barometric pressure (P_B) decreases from 760 mmHg at sea level to 272 mmHg on the Everest summit, and inspired pressure of oxygen (P_IO_2) decreases from 149.7 mmHg to 47.1 mmHg *(Grocott-NEJM-2009)*. In the course of the 1981 American Medical Research Expedition to Mt. Everest (AMREE), Dr. Christopher Pizzo collected exhaled gas samples on himself on the summit *(West-Pizzo-JAP-1983)*. Dr. Pizzo's alveolar PO_2 was 28 mmHg and his alveolar PCO_2 was 7.5 mmHg at the summit. Audio tape recordings by Dr. Pizzo on the summit revealed that he was panting for breath between every two or three words *(West-JWM-1993)*. Subsequent investigators have corroborated these observations *(Grocott-NEJM-2009)*.

Studies of gas exchange on the summit of Mt. Everest are insightful at several levels. Dr. Pizzo and other climbers were able to tolerate profoundly low PO_2 levels as a result of slow gradual

acclimatization: he had been exposed to high altitude for 77 days before he took his summit samples *(West-JWM-1993)*. Un-acclimatized persons lose consciousness within 2-3 min if suddenly exposed to levels of ambient hypoxia equivalent to those at 8400 m (27,559 feet): P_B 272 mmHg, P_IO_2 47.1 mmHg, P_AO_2 30.0 mmHg *(Grocott-NEJM-2009)*.

An observation of fundamental importance from studies at extreme attitude is recognition that a mountaineer's survival is critically dependent on the ability to achieve extremely high levels of alveolar ventilation. The graph below shows a plot of alveolar PO_2 and alveolar PCO_2 in climbers as they ascend from sea level (top right) to the Everest summit (bottom left) *(West-FP-2019)*.



Alveolar PO_2 decreases because of the decrease of oxygen in the air around the climber consequent to fall in barometric pressure. Alveolar PCO_2 decreases as a result of the climber's increase in alveolar ventilation (blowing off CO_2). After an altitude of about 7000 m (22,966 feet) has been reached, associated with PCO_2 of about 20 mmHg, climbers exhibit no further decrease in the alveolar PO_2. As seen on the figure, alveolar PO_2 is defended at a level of about 35 mmHg *(West-JWM-1993)*. This defense is crucially dependent on extreme increases in alveolar ventilation as altitude rises causing progressive lowering of alveolar CO_2 to make room for alveolar O_2. For a climber to survive on Everest, he or she needs to be able to increase alveolar ventilation to an extent that drives PCO_2 down to around 8 mmHg (normal, 35-45 mmHg) and thus preserve the alveolar PO_2 at the very low but viable level of about 35 mmHg *(West-JWM-1993)*. In healthy non-acclimatized subjects, the minimum PaO_2 that avoids cerebral hypoxia (inadequate brain oxygen) is 37 mmHg (individual variation is considerable) *(Nunn-p474)*. The doyen of pulmonary gas exchange, John B West, MD (b 1928), emeritus professor of physiology, University of California San Diego, calculated that Dr. Pizzo needed to increase his alveolar ventilation 6-fold in order to achieve his recorded alveolar PCO_2 of 7.5 mmHg on the Everest summit *(West-JWM-1993)*. Additional evidence emphasizing the crucial importance of hyperventilation for survival is the observation that climbers who have a low ventilatory response to hypoxia have a lower rate of

success in reaching the summit and tolerating extreme altitudes *(West-JWM-1993; Schoene-JAP-1984)*.

The above discussion of gas exchange on Everest is confined to mountaineers who survived the climb. Mountaineers are a selected group of extremely healthy individuals, yet the risk of hypoxia and physical challenges pose a real risk of death (especially on descent). The danger of hypoxemia is highlighted by greater number of deaths among mountaineers not using supplemental oxygen than in mountaineers using supplemental oxygen: 8.3% v 3.0% for Mt. Everest and 18.8% v 0.0% for K2 (8,611 m, 28,251 feet, second-highest mountain after Everest) *(Huey-JAMA-2000)*. Most climbers today begin using supplemental oxygen at about 7200 m (23,600 feet; at Everest Camp 3, 7100 m (23 294 feet), P_B is 317 mmHg and P_IO_2 is 56.5 mmHg *(Lankford-WEM-2021; Grocott-NEJM-2009)*. The Todeszone (Death Zone) refers to the perilous risk at mountains above 7800-7900 m (25,500–26,000 feet) *(Lankford-WEM-2021)*.

Defense attorney Ms. Lindsay Gowin asked defense medical expert Dr. Stephen Bird to "explain the amount of carbon dioxide that a person can ventilate off in exhalation." On page 149 of his deposition, Dr. Bird answered:

> "You're able to physiologically, you're able to blow off CO_2 down to no more than 15, level of 15. And lower than that really isn't physiologically possible…. Once you get to around 15, there's no more ventilation that you can."

This statement is wrong. Dr. Pizzo recorded an alveolar PCO_2 of 7.5 mmHg on the summit of Everest *(West-JWM-1993)*. Levels of PCO_2 lower than 15 mmHg are not confined to mountaineers. Mazzara et al *(AJM-1974)* reported 25 ICU patients with PCO_2 levels of 10 to15 mmHg. Rodriguez et al *(Ann-Neurol.-1982)* reported PCO_2 9 mmHg in a patient with a brainstem tumor. Throughout his deposition, Dr. Bird makes confident assertions of physiological occurrences in Mr. Timpa even when – as in this example – they fly in the face of long-known medical facts published in the scientific literature many years ago.

In the discussion section of the Michalewicz-Chan article *(Michalewicz-Chan-AEM-1997)*, the authors state "we conclude that PMRP [Prone Maximal Restraint Position] and prone positioning with moderate weight force on the back do not in and of themselves restrict metabolic or ventilatory demands to any clinically important degree. As such, factors other than isolated ventilatory failure should be considered when evaluating deaths occurring in the setting of restraint in the field" *(Michalewicz-Chan-AEM-1997)*. Dr. Chan argues that a 30% decrease in pulmonary function is not clinically significant because it does not produce a decrease in SpO_2 on pulse oximetry (an insensitive yardstick in healthy subjects), but misses the point that a detainee restrained by a police officer kneeling on his back needs to increase alveolar ventilation 6-fold in order to blow off CO_2 and make room for alveolar O_2, and thus avoid fatal hypoxemia *(West-JWM-1993)*. Such an increase in alveolar ventilation is impossible during restraint because chest compression impedes the ability of the respiratory muscles to expand the thoracic cavity *(Tobin-Clinics-Chest-Med-1988)*, and, thus, the detainee is unable to maintain even a normal level of alveolar ventilation, let alone increase it 6-fold. Dr. Chan's failure to consider the need for alveolar hyperventilation to defend against fatal hypoxemia (during restraint) undermines his claim that weight on the back does not restrict ventilation to any clinically significant degree. Research studies of gas exchange in mountaineers highlight the potentially fatal consequences of Dr. Chan's flawed assertions.

In addition to the three fatal flaws discussed above, there are other problems with the design and interpretation of the Chan experiments. The studies were conducted in healthy volunteers who knew they could withdraw at any point – a situation antithetically different from a person restrained by police and fighting for his life. Of 30 young people recruited for the Michalewicz-Chan experiment, valid data could not be collected in 3 subjects – 2 subjects were "frightened of being restrained" and "were psychologically unable to tolerate restraint" *(Michalewicz-Chan-AEM-1997)*. Subjects were placed in the Prone Maximal Restraint Position (PMRP) and requested to struggle vigorously for 60 sec. Regarding 27 participants in this portion of the study, the authors remarked "The struggle was physically difficult for subjects. In spite of continued verbal encouragement, the intensity of movement was visibly waning in all subjects by the end of the 60-sec trial" *(Michalewicz-Chan-AEM-1997)*. Sixty seconds is less than 10% of the duration of restraint to which Mr. Timpa was subjected.

One of the most influential champions of Dr. Chan's research has been Dr. Vincent Di Maio (1941-2022), who served as Medical Examiner in Dallas (1972-81) before becoming Chief Medical Examiner of Bexar County. With his wife, Dr. Di Maio published a book "*Excited Delirium Syndrome: Cause of Death and Prevention,*" which he

> "dedicated to all law enforcement and all medical personnel who have been wrongly accused of misconduct in deaths due to excited delirium syndrome" *(Di Maio-2005-page v)*.

Dr. Di Maio claimed that belief in positional asphyxia "involves suspension of common sense and logical thinking" and that Dr. Chan's 1997 report dealt a "death blow" to the theory of positional asphyxia *(Di Maio-2005-p35, p23; Chan-Clausen-AEM-1997)*. TASER purchased 1,000-1,500 copies of Dr. Di Maio's book and distributed free copies at conferences of medical examiners and police chiefs *(McGuinness-Lipsedge-2022; PHR-2022)*. On page 176 of her deposition, Medical Examiner Dr. Emily Ogden describes having a 90-min meeting with "Lindsay" (Dallas City Attorney Ms. Gowin) and "Tatia" (Dallas City Attorney Ms. Wilson) before her deposition, and that she read Dr. Di Maio's book after the meeting with the attorneys.

Throughout his book, Dr. Di Maio referred to Dr. Chan's claim that while restraint might produce a decrease in lung function, the consequences are not clinically significant. On page 24 of the book, Dr. Di Maio discussed Dr. Chan's study of changes in pulmonary function tests (PFT) following restraint and exercise. Dr. Di Maio noted:

> "However, the PFT changes while statistically significant were not clinically relevant…. Most importantly, there was no evidence of hypoxia in the restraint position after exercise with no evidence of hypercapnia either during exercise or in restraint."

Dr. Di Maio failed to consider the insensitivity of pulse oximetry at detecting huge 40-mmHg decreases in PaO_2 in healthy people (see earlier discussion on effects of shift in the oxygen dissociation curve on pulse oximetry). Dr. Di Maio also failed to recognize that an increase in PCO_2 is a terminal event in the setting of hypoxemia, as demonstrated by research on Mount Everest (discussed above). On page 27 of the book, Dr. Di Maio stated:

> "The medical profession often ignored the work of Chan et al., although this work had put a major crimp in lawsuits against police departments where claims of positional asphyxia were made.[28] That the legal profession accepted the work of Chan et al. while the medical profession still espoused theories that had been proved wrong by scientific testing is

> fascinating. While some individuals acted as if Chan et al.'s work had never been performed, others challenged it on illogical grounds."

Judgments about the credibility of research findings are based on a rigorous analysis of methodology of experiments and understanding of scientific context, rather than on acclaim from the legal profession. On page 36 of the book, Dr. Di Maio discussed another Chan publication:

> "Chan et al.[16] conducted a series of experiments to determine if placing an individual prone in the hog-tied position, following strenuous exercise, produced restriction in ventilation such that there was impairment in oxygenation of blood. They found that while this resulted in restrictive pulmonary functioning as measured by pulmonary function tests (PFT), the changes were not clinically relevant. There was no evidence of hypoxia in the restraint position after exercise, as well as no evidence of hypercapnia either during exercise or in restraint."

On page 69 of the book, Dr. Di Maio stated:

> "For many years, it was believed that death in excited delirium syndrome was due to positional or restraint asphyxia. The problem with this hypothesis is that experiments by Chan et al have essentially disproved this hypothesis. There is no scientific evidence that restraint results in hypoxia."

Dr. Di Maio never considered the numerous methodological physiology flaws in the above discussed experiments that render Dr. Chan's conclusions null and void.

In his deposition, Dallas Chief Medical Examiner, Dr. Jeffrey Barnard, made several statements that resonate with the views of Dr. Chan and Dr. Di Maio. On page 133 of his deposition, Dr. Barnard remarks that a knee on the back prone can cause "some compromise of respiration," but adds the caveat

> "Whether it's clinically significant becomes a different issue."

On page 161 of his deposition, Dr. Barnard refers to research studies showing "some compromise of respiration," but again dismisses the compromise by adding

> "It just is not clinically significant."

Lower on page 161, Dr. Barnard states

> "And if it did play – the compromise which is normally not clinically significant – excuse me – would not be clinically significant."

On page 162, Dr. Barnard discusses the Michalewicz-Chan study, adding "So that in a prone position, bound, and some hogtied, there was compromise. It just wasn't clinically relevant." Later on page 162, Dr. Barnard comments

> "So in this case, he's not hogtied, so he's not bowed like they do with that, that it should not have clinical significance."

On page 198 of his deposition, Dr. Barnard states

> "I said I can't tell whether the mechanical compression contributed – was clinically significant or not."

On page 199, Dr. Barnard states

> "As I said earlier it's shown in these studies on healthy people that there is some respiratory compromise. It just isn't clinically significant"

Then adding

> "It would not be clinically significant in this gentleman [Mr. Timpa]."

In 9 different locations in his deposition, Dr. Barnard communicated that he accepts Dr. Chan's claim that decreases in lung function consequent to kneeling on the back of a prone subject are not

of a magnitude to be clinically significant, and never contemplated the level of ventilation required in physiological settings of increased ventilatory demands. On page 119 of his deposition, Dr. Barnard refers to Dr. Guyton's textbook, claiming

> "It's kind of considered probably the best physiology book."

I used Guyton's text as a second-year medical student, 1970-71, and, while satisfactory for students, Guyton's text does not provide the breath or depth of understanding or the nuances and complexities of respiratory physiology required to appreciate the glaring methodological flaws in Dr. Chan's studies.

In the course of his deposition, defense medical expert, Steven Bird, MD, also conveys an uncritical acceptance of Dr. Chan's findings. On page 40 of his deposition, Dr. Bird states

> "Generally, the Chan article or articles, I only cited the one, looking at that demonstrate that there is no chronically (*sic*, clinically) significant changes in restraint versus weight versus another position."

Later on page 40, Dr. Bird states

> "that is with the limitations of a controlled study that they there's no clinically significant change in respiratory dynamics."

On page 78 of his deposition, Dr. Bird states

> "There is non-clinically significant changes in some respiratory dynamics with weight, but it takes hundreds of pounds in order to really affect somebody's to clinically significant affect someone's breathing."

On page 123, Dr. Bird states

> "The scientific evidence is that this will not adversely, clinically, significantly adversely affect his breathing."

On page 123, Dr. Bird states

> "They looked at ventilation parameters with up to 103 kg and I believe they found no statistically significant difference with the weight applied up to 103 kg."

As with Dr. Barnard, Dr. Bird fails to consider the level of respiratory reserves needed in physiological settings of increased ventilatory demands.

Defense expert, Mark Kroll, PhD, likewise communicates uncritical acceptance of Dr. Chan's findings during his deposition. On page 161, Dr. Kroll states

> "And then we had the work from UCSD with 225 pounds on the back and noticed that there was no clinically significant restriction of breathing."

On page 129 of his deposition, Dr. Kroll states

> "People have gone to greater extremes where they've had the restraint behind the back and then tied the ankles. And it's been shown that has no clinical significant reduction in ventilation."

When Mr. Henley pointed out that there was a 15% reduction in lung volumes in a Chan study, Dr. Kroll responded (page 129)

> "Yes from maximum voluntary ventilation. But we have such an enormous reserve. We have a 30:1 reserve."

On page 131, Dr. Kroll adds

> "So, if that is reduced down by 15%, that's still way more than you need."

Dr. Kroll fails to recognize that when faced with increased ventilatory demands, such as at high altitude or being restrained by police, a 15% decrease in lung volume may make the difference

between life and death. On page 151, Mr. Henley asked Dr. Kroll about the 30% decrease in MVV observed in the Michalewicz-Chan study, and Dr. Kroll responded:

"The significance is there was no significance."

D. **Dr. Reay's "retraction" of research findings**

In his expert report of November 1, 2019, Dr. Mark Kroll, PhD, discussed the research findings of Dr. Reay, claiming that Dr. Reay had retracted his previous statements on positional asphyxia. At Dr. Kroll's deposition, Mr. Henley inquired "You think that Dr. Reay completely conceded everything that he found in the beginning?" and Dr. Kroll answered: "That's my understanding" (page 145).

Steven Bird, MD, stated at his deposition: "But the positional asphyxia as initially advocated by Reay in 1992, Doctor Reay recanted and said that isn't a thing" (page 47). Plaintiff attorney Mr. R. Lane Addison inquired "You said that Reay recanted his study?" and Dr. Bird answered: "Correct or recanted the occurrence of positional asphyxia" (page 50). On page 80 of his deposition, Dr. Bird claimed: "Dr. Reay says in court that this doesn't happen," and, on page 82, Dr. Bird added "I think he's [Dr. Reay] disavowed the concept of positional asphyxia."

While serving as Chief Medical Examiner of King County, Washington between 1975 and 1999, Donald T Reay, MD (1937-2018) published several articles related to neck holds, positional asphyxia, death during law enforcement transport, and investigating and certifying deaths in police custody *(Reay-AJFMP-1982; Reay-AJFMP-1992; Reay-CLM-1998; LukeReay-AJFMP-1992)*. In 1996, Dr. Reay served as plaintiff expert witness in *Price v. County of San Diego*, and emergency room physician Tom S Neuman, MD, colleague of Dr. Chan, served as defense expert witness. This lawsuit instigated the subsequent substantial body of research by Dr. Chan's group into the effects of restraint, and, in the first of their many publications on this topic *(Chan-AEM-1997)*, Chan et al acknowledged that the research was supported by a grant (No. 94-1974R) from the County of San Diego (defendant in the Price trial). In a recent report, Dr. Weedn *(Weedn-JFS-2022)* of the Office of the Chief Medical Examiner, District of Columbia, Washington remarked: "After a lawsuit was launched against the County of San Diego for the death of Daniel Price in 1994 [24], the County funded a medical group at the University of California at San Diego to perform experimental studies involving weights placed on young healthy subjects in the prone position. That San Diego group and their colleagues published several reports concluding that the effect on ventilation was clinically insignificant." The research findings of Dr. Chan's group were judged to be a critical factor in the successful defense of the County of San Diego in the Price trial.

Experts defending police in lawsuits, in addition to Dr. Kroll and Dr. Bird, claim that Dr. Reay had retracted his previous views on positional asphyxia. In September 1999, Dr. Reay and Dr. Howard published a report entitled "Restraint position and positional asphyxia," in the *American Journal of Forensic Medicine and Pathology* *(Reay-Howard-AJFMP-1999)*, in which they commented on an article by Drs. Chan, Vilke and Neuman *(Chan-AJFMP-1998)*. In their commentary, Dr. Reay and Dr. Howard wrote "We acknowledge that the hogtied position should be viewed as an inherently neutral position, and although there is a measurable change in ventilatory function, there is no significant physiological consequences in normal people." This statement refers solely to the hogtied position, yet, for whatever reason, experts defending police in lawsuits claim that Dr. Reay had retracted his previous views on positional asphyxia.

In their September 1999 commentary, Dr. Reay and Dr. Howard pointed out:

> "We still have concern regarding deaths that occur during restraint. From the work of Chan, we now know that the hogtied position should not produce serious physiological consequences. However, during street restraint maneuvers, the totality of events must be considered. In the process of rendering a person helpless to handcuff him or her in a prone position, the involved officers may be required to pile on the suspect, pinning the person to the ground with the partial or full weight of the officers and thus compressing and restricting ventilatory function. The physical condition of the person and the circumstances of restraint can make a difference, and each case must be evaluated with a careful consideration of events to identify respiratory interference during and after the takedown person is restrained" *(Reay-Howard-AJFMP-1999)*.

Dr. Reay became aware that his San Diego testimony was being misrepresented, and he sent a notarized statement to Ms. Charly D. Miller, which contained the following:

> "I readily acknowledged the value of these studies in the San Diego case of "Price vs. San Diego" which had many other features besides hog-tying in the restraint maneuvers used to control the victim. This has since been presented in law enforcement publications as my retraction of positional asphyxia as a cause of death, with particular reference to hogtying. Such is not the case! I still maintain that there are risks and hazards to restraint maneuvers including hog-tying and each case must be evaluated to assess the presence or absence of respiratory restriction in the light of the method of restraint. …
> The point is that street deaths are much different than controlled investigations. If 14% respiratory restriction by hog-tying is not viewed as clinically significant in normal people, it has to be evaluated in the context of the event where it may be significant" *(Reay-Miller-1998)*.

These commentaries by Dr. Reay are easily retrieved through the Internet, and it is difficult to understand why experts in legal cases defending police continue to misrepresent Dr. Reay's stance on positional asphyxia.

E. **Excited delirium syndrome and misattributions**

In his expert report and during his deposition, Dr. Bird claims that Mr. Timpa's death was caused by excited delirium syndrome. Excited delirium syndrome is frequently offered as a reason for deaths in persons detained by police, yet proponents are unable to provide a credible explanation for how this syndrome causes a person to die. A number of putative processes have been invoked, such as abnormal dopamine function, heat-shock proteins, or myocardial channelopathy, but proponents have not presented credible scientific data for how these processes are the cause of sudden death among detainees in police custody *(DiMaio-Book)*.

On page 30 (line 8) of his deposition, Dr. Bird stated:

> "Excited delirium has been known for over150 years and it's been known as other things such as Bell's Mania."

This statement is factually incorrect. Dr. Luther Bell was superintendent of the McLean Asylum for the Insane (a division of the Massachusetts General Hospital) between 1837 and 1858. In 1849,

Dr. Bell reported in the *American Journal of Insanity* that he had seen 40 cases of a grave form of mania across 12 years *(Bell-1849)*. He reported details of 10 case histories, involving 6 men and 4 women. The case histories exemplified mania of sudden onset combined with disorientation, confusion, and fluctuating consciousness characteristic of delirium. Three quarters of Dr. Bell's patients died, and autopsy did not reveal a convincing underlying cause. The duration of delirious mania in 6 patients (described in case histories) ranged from 12 days to 8 weeks (average duration, 25.5 days) *(Bell-1849)*. Dr. Bell's description of death occurring 25 days after hospital admission is diametrically different from individuals reported to have excited delirium who exhibit mental distress for only a matter of hours and die minutes after the application of restraint *(DiMaio-Book)*.

In the 170 years following Dr. Bell's article, few authors have reported on this condition. In 1980, Thomas Bond, MD, (Department of Psychiatry, McLean Hospital, Harvard), reported 3 case histories of patients with "acute delirious mania" distinguished by acute onset of irritability, insomnia, emotional withdrawal, features of hypomanic or manic syndrome (as defined by DSM-III criteria), and development of signs and symptoms of delirium *(Bond-AGP-1980)*. None of Dr. Bond's patients died. In 1999, Max Fink MD (Department of Psychiatry, SUNY Stony Brook), described 14 patients with "delirious mania," who featured acute onset of excitement, grandiosity, emotional lability, delusions, insomnia, disorientation, and altered consciousness *(Fink-BD-1999)*. None of Dr Fink's patients died. The clinical descriptions of acute delirious mania by Dr. Bond, Dr. Fink (and Dr. Bell) are completely different from the clinical description of detained individuals reported to have excited delirium syndrome *(DiMaio-Book)*.

No other example arises in the broad expanse of medicine where physicians base present-day scientific claims on a 170-year-old poorly documented clinical case series. The precise mechanism of death of Dr. Bell's patients is unknown. Subsequent reports of unexpected deaths in psychiatric patients document a high rate of institutional neglect *(Derby-PQ-1933; Davidson-AJP-1934; Shulack-PQ-1938)* and missed fatal diagnoses *(Regestein-JAMA-1977; Funayama-PM-2018)*. Many important medical disorders now recognized as causes of unexpected death in psychiatric patients *(Derby-PQ-1933; Davidson-AJP-1934; Shulack-PQ-1938; Regestein-JAMA-1977; Funayama-PM-2018)* had not been described in 1849. Descriptions of venothromboembolism and pulmonary embolism were first presented in the late 19th century *(McFadden-Ochsner-2002)*, histological methods for the diagnosis of myocardial infarction were introduced around the same time *(Jennings-Circ-Res-2013)*, and the sphygmomanometer necessary for documentation of blood pressure in the diagnosis of septic shock was not invented until 1896 *(O'Brien-JHH-1994)*. These and many other diseases could not have been detected in the poorly documented autopsies performed on Dr. Bell's patients in the 1840s. To equate death of detainees in police custody in the 21st century to the nature of death in Dr. Bell's patients of the 1840s far exceeds the bounds of scientific credibility.

Two groups of physicians make a diagnosis of excited delirium syndrome more than other doctors: medical examiners (pathologists) and emergency room physicians. Because every patient seen by a medical examiner is already dead, research investigations into the lethality of some medical condition are especially vulnerable to test-referral bias, a treacherous hazard in the design of scientific experiments *(Tobin-ICM-2006)*. Without explicit documentation of deliberative steps taken to ensure that test-referral bias did not occur, the scientific validity of medical-examiner conclusions on the mechanism of death in excited delirium syndrome is subject to major doubt *(DiMaio-Book; Stephens-AJFMP-2004)*.

Although articles on excited delirium are frequently written by emergency room physicians, such as Dr. Bird, these clinicians see relatively few patients with the general condition of delirium, as contrasted with intensive care physicians. About ⅓ of ICU patients exhibit delirium, and ½ to ¾ of patients on a mechanical ventilator exhibit delirium *(Wilson-Ely-Delirium-Nat-2020)*. In contrast, delirium is observed in 0.59% of patients seen in an emergency room *(Miner-AEM-2018)*.

Delirium is triggered by multiple factors in critically ill patients, such as sepsis, stroke, liver failure, drug use, surgery, and psychological stress *(Wilson-Ely-Delirium-Nat-2020)*. Patients exhibiting delirium may recover completely (usual outcome), transition to another organic brain syndrome or a non-organic mental disorder (schizophrenia, a rare event), or die *(Lipowski-1980-Kaplan-Textbook-p1368)*. If a patient dies during a bout of delirium, the underlying organic cause of death is expected to be uncovered by clinical assessment or autopsy (such as sepsis and its source) *(Lipsedge-MSL-2016)*. In other words, hospital and ICU patients die *with* delirium – not *because of* delirium.

Major medical organizations, such as the American Medical Association (AMA) and the World Health Organization (WHO), advise physicians not to employ the term excited delirium syndrome, arguing that it is not a sound medical diagnosis. The term, however, is still employed by some attorneys. Moreover, courts of law perform a gatekeeper function regarding the "admissibility" of expert testimony, based on whether the methodology of an expert is scientifically valid and can properly be applied to the facts in a case *(Daubert standard)* *(PHR-2022)*. Many courts (i.e., the presiding judge) have ruled that expert testimony on excited delirium syndrome should be *admitted* as *evidence* at trial—the onus then being on the opposing attorney to argue against the *persuasiveness* of the theory as credible evidence *(PHR-2022)*. When plaintiff attorneys have sought to exclude testimony on excited delirium syndrome, courts have pointed to three communities that generally accept it as a diagnosis: the American College of Emergency Physicians (ACEP), the National Association of Medical Examiners (NAME), and police departments *(PHR-2022)*.

In 2009, an ACEP Task Force published *The White Paper*, which made two major claims: excited delirium syndrome is a real medical disorder, and its diagnosis can be made when patients display 6 of 10 listed signs *(DeBard-2009)*. In June 2021, a new Task Force published a new report, stating that ACEP no longer recommended use of the term excited delirium *(Cole-ACEP-2021)*. In 2004, the National Association of Medical Examiners (NAME) published a Position Paper on certification of cocaine-related deaths and the role of excited delirium syndrome in causing such deaths *(Stephens-AJFMP-2004)*. This Position Paper provided a foundation that fostered the attribution of sudden death of detainees in custody to excited delirium syndrome by a growing number of medical examiners. In 2022, NAME president, Dr. Kathryn Pinneri, reversed its previous position *(PHR-2022)*, stating that

> "as an organization [NAME has] not formally 'recognized the condition [excited delirium syndrome] as a diagnosis.' The NAME Position paper on the Certification of Cocaine-Related Deaths *(Stephens-AJFMP-2004)* is no longer current and therefore does not reflect our position at this time" *(PHR-2022)*.

Critics of excited delirium syndrome focus primarily on the validity of the diagnosis, citing organizations such as the AMA, WHO, and the American Psychiatric Association (APA) and other bodies who proclaim that it is not a legitimate diagnosis. While I agree with this criticism, the labeling of the entity is not its greatest problem. Instead, the fundamental problem is the inability of proponents of excited delirium syndrome to explain how a non-organic (psychiatric-mental) disorder can cause somebody's death. Only two psychiatric conditions are known to cause death: anorexia nervosa, a chronic condition where death consequent to self-starvation is easy to understand, and lethal catatonia, an extremely rare psychiatric disorder, formerly viewed as universally fatal, but, given the survival of many present-day patients, the disorder is currently termed malignant catatonia *(Castillo-AJP-1989; Daniels-NCN-2009)*. Writing on death of patients with delirium, the noted London psychiatrist, Dr. Maurice Lipsedge, emphasizes

> "the cause of the patient's death is not their psychiatric disorder or their agitated behaviour, but a grave underlying medical condition."

Lipsedge adds that

> "in the absence of an underlying physical cause for an acute behavioural disturbance," a claim that "delirium" is the cause of death becomes self-contradictory" *(Lipsedge-MSL-2016)*.

The distinction between organic disorders and non-organic (functional) psychiatric disorders is crucial in understanding causation of death in persons who are suspected to have excited delirium syndrome or a related condition. By definition, there is no identifiable underlying organic pathological disease in patients with a primary functional psychiatric condition *(Lipsedge-MSL-2016)*. Dr. Barnard, Dallas Chief Medical Examiner, is apparently unaware of this crucial distinction. On page 126 of his deposition, Dr. Bernard stated:

> "someone has an organic disease such as schizophrenia."

Schizophrenia is emblematic of functional psychiatric disorders and is not an organic disease.

The most detailed research study on excited delirium syndrome is that conducted by Dr. Strömmer and colleagues and published in August 2020 – subsequent to the expert reports and depositions in the Timpa case *(Strömmer-FSMP-2020)*. After searching the world literature, these investigators identified 168 published cases of excited delirium syndrome. Of the total 168 cases, 104 died (62%) and 64 survived (38%). Among the 104 deaths, some form of restraint was documented in 90%, and there was documentation of no restraint in only two deaths (2%). Based on their rigorous scientific analysis, Dr. Strömmer and colleagues concluded that use of restraint was the key factor in deciding whether or not somebody with excited delirium lived or died *(Strömmer-FSMP-2020)*.

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ACEP Task Force Report on Hyperactive Delirium with Severe Agitation in Emergency Settings. Approved by the ACEP Board of Directors, June 23, 2021 [Jon B. Cole, MD Richard P. Dutton, MD, MBA James R. Gill, MD Nicole R. Jackson, MD, MPH Louise W. Kao, MD Kurt C. Kleinschmidt, MD Douglas F. Kupas, MD Eric J Lavonas, MD, MS David Manyura, BSN, RN Lewis S. Nelson, MD Gerald F. O'Malley, DO Samuel J Stellpflug, MD Andrew Stolbach, MD, MPH Shawn Varney, MD Yanling Yu, PhD]

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Appendix #1

Flash drive containing the following material:

Third Amended Complaint;
Custodial Death Report;
Death Certificate;
Autopsy and all exhibits;
Incident reports;
Timeline;

Experts' Reports and depositions:
Kimberly Collins, M.D.-Plaintiffs/Intervenor
Michael Lyman, Ph.D.-Plaintiffs/Intervenor
Mark Kroll, Ph.D.-Defendants
Steven Bird, M.D.-Defendants
Craig Miller, B.A.-Defendants;

Dallas Police Department Defendants' Depositions:
Dustin Dillard
Domingo Rivera
Raymond Dominguez
Kevin Mansell

Dallas Police Department Command Staff:
Rick Watson;
William Griffith;
Chandra Griffith;

Dallas Fire and Rescue:
Curtis Burnley;
James Flores;

Dallas County Medical Examiner's Office:
Emily Ogden, M.D.;
Jeffrey Barnard, M. D.;

Recordings:
911 recordings;

Videos:
Body cams:
Dillard body cam;
Vasquez body cam;
Rivera body cam;

Appendix 2

Timed Entries of Events

Time			Dillard Body				Vasquez Body	
Vasquez Camera	Dillard Camera	Rivera Camera	Left Knee	Right Knee	Left Hand	Right Hand	Left Knee	Other
01:27	00:49				Turning Mr. Timpa prone	Turning Mr. Timpa		Turning Mr. Timpa
01:28	00:50				One of three hands on Timpa's torso	One of three hands on Timpa's torso		L. Hand is one of three hands on Timpa's torso
01:29	00:51		Timpa's back		Left Torso	Possible Ground	Left Torso	Removes hand
01:35	00:57		"		"	Ground	"	"
02:06	01:28		"		"	Off Timpa	"	"
02:14	01:36		"	Timpa's back	"		"	"
02:26	01:48		"	"	"	Right Torso	"	"
02:29	01:51		"	"	Raised	"	"	"
02:30	01:52		"	"	Left Torso	"	"	"
02:44	02:06		"	"	"	Ground	"	"
02:53	02:15		"	"	"	Right Torso	"	"
03:28	2:50		"	Off	"	"		"
03:35	02:57		"		"	"	"	"
04:07	03:29		"		"	"	Off	"
04:24		01:59	"		"	"	"	"
05:28	04:50		"		Assists with cuffs	"	"	"
06:04	05:26		"		Lifting Timpa's left arm	"	"	"
07:13	06:35		"		Off	"	"	"
07:17	06:39		"		Lifting Timpa's left arm	"	"	"
07:19	06:41		"		"	"	"	Has removed handcuffs
07:30	06:52		"		Off	"	"	"
07:36	06:58		"		Repositioning	"	"	"
07:46	07:08		"		Lifting Timpa's left arm	"	"	"
08:08	07:30		"		Left Torso	"	"	"
08:28	07:50		"		"	Raised	"	"
08:34	07:56		"		Raised	"	"	"
08:38	08:00		"		Left Torso	Ground	"	"
09:24	08:46		"		"	Raised	"	"

09:26	08:48		"		"	Likely ground	"	"
09:28	08:50		"		"	"	"	Left hand on Left Torso
09:56	09:18		"		"	"	"	"
10:05	09:27		"		"	"	"	"
10:16	09:38		"		"	"	"	"
10:34	09:56		"		"	Possible R. Torso	"	"
10:49	10:11		"		"	"	"	"
11:08	10:30		"		"	"	"	"
11:19	10:41		"		"	"	"	"
13:08	12:30		"		"	Right Torso	"	"
13:29	12:51		"		"	"	"	Left hand off, right hand left torso
14:03	13:25		"		Gives Timpa a shake	"	"	"
14:18	13:40		"		Gives Timpa a shake	"	"	"
14:29	13:51		Removed		"	"	"	"
14:32	13:54		Applied		"	"	"	"
14:40	14:02		"		"	"	"	"
15:07	14:29		"		"	"	"	Begins standing, initially keeps right hand on Timpa
15:09	14:31		"		"	"	"	Removed self
15:15	14:37		"		Removed	Removed	"	"
15:37	14:59		Removes self from Timpa		"	"	"	"
16:00	15:22		Timpa placed upon gurney					